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Table of Contents

PROBLEMS IN THE WORKING OF A CHILD WELFARE SCHEME IN THE COMMUNITY.[1]

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FROM earliest times it has been realized that the health of the community is all important. Preventive medicine in primitive communities takes the form of religious rites, aiming at the propitiation of a deity, and so in this stage of a nation's development the practice of medicine is in the hands of a priesthood. The Greeks did much to disentangle the art of healing from a maze of religion, superstition and witchcraft, and may be said to have laid the foundation of the profession of medicine as we know it. The process of evolution has, however, been a slow one, and many difficulties have had to be overcome, increasing knowledge and changing conditions demanding continual adjustments.

In every age the growth of knowledge has seemed considerable, but in our time it has been so phenomenal that the difficulty of integration has become acute. Every section of our work is now so amplified that it has become impossible to be an expert in all branches, and a great impulse towards specialization has been the result.

With regard to child welfare this evolution in the last hundred years or so has been particularly noticeable, what might be described as a renaissance having taken place. This can be appreciated by looking back into the seventeenth and eighteenth centuries. There one sees an enormously high infant mortality rate, the employment of child labour and an unhygienic, uneconomic, not to say inhuman, state of affairs.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 26, 1931.

Children's hospitals are so much a part of our daily life that it seems strange to think that the first to be founded in England was that of Great Ormond Street, in 1852—not eighty years ago. Laws for the protection of child life were enacted in England only so recently as 1872, and almost the first "infant consultations" to be opened were those of Professor Budin, of Paris, in 1892.

In the seventeenth and eighteenth centuries artificially fed babies seldom lived to grow up, the oft-quoted statistics of the Dublin Foundling Hospital surely marking the peak of infant destruction, namely, the survival of only 45 babies out of 10,272 admitted to the institution during twenty-one years.

In our time the child welfare movement has grown apace. There are children's hospitals in all large cities, infant consultations have become baby clinics and have spread all over the world, child labour is largely a thing of the past, and education is given freely to all. Popular opinion has changed to such an extent that instead of criminal neglect of child life there has developed an almost embarrassing sentimentalism.

No one can doubt that the establishment of child welfare centres and the work done in connexion with them have been factors of importance in producing a lower infant mortality rate of late years. In South Australia the infant mortality rate in the years 1870-1874 was 146·76‰; in 1900-1904, 91·95‰; in 1929, 40·88‰; and in 1930, 48·28‰. The year 1929 was the lowest on record in Australia. The rate this year has so far shown an improvement on these figures.

State government and municipal bodies have been impressed with the importance of the work done by the child welfare organizations and are willing to spend large sums of money annually on their support. New South Wales spends about £41,000, Victoria about £25,000, Queensland £12,000, and Western Australia and Tasmania between £1,000 and £2,000 a year. In this State the figure is £5,500.

For the year ending July 31, 1931, the total attendances at our centres numbered 93,176, and 36,396 visits were paid by the nurses.

Tonight my special concern is to bring before you some of the problems involved in the working of a child welfare organization in this community. The Mothers' and Babies' Health Association was established in Adelaide under the name of "School for Mothers" in the year 1909. It had a humble beginning in a cottage in Franklin Street, where mothers were encouraged to bring their babies to be weighed and to receive advice regarding feeding and general hygiene. The movement has grown and there are now thirty-five metropolitan and six country centres. The essentials for the working of each district are:

1. A committee which undertakes the financial and general management.

2. A building to be used as a depôt.

3. Certain equipment, such as accurate scales, apparatus for demonstration purposes, educational material of various kinds.

4. Nurses who have undergone special training in child welfare in addition to general and obstetric training.

5. Medical officers, at present honorary, for work with children or mothers at the centres.

6. The mothers and babies, also children up to six years of age.

The whole group of centres is united under a chief centre, with its matron in charge of the whole nursing staff. At present with the matron there are twenty nurses.

The work of a centre consists in:

1. The visiting by the nurse of new-born babies whose names are obtained from the Registrar of Births.

2. Regular weighing of the babies that come to the centres.

3. The education of the mothers as to correct methods of feeding and general hygiene.

4. At certain centres a medical practitioner is in attendance every fortnight for the purpose of examining children and referring them in cases of ill health to their own medical attendant or to the Children's Hospital, as their means permit.

5. At one centre an antenatal consultation has been established and any abnormal conditions found are referred to the appropriate quarter for treatment. When possible, others will be established.

6. Demonstrations are given in the bathing of babies, sets of clothes are kept and instruction is given to the mothers on their use and construction.

7. Test feeds are carried out as a guide to correct feeding. The baby is weighed before and after nursing, and so the amount of milk taken is known. A single feed, or better, a series of feeds, thus measured, gives a very good idea of what the baby is getting, and consequently if there is over or underfeeding present. A medical practitioner wanting this information has only to refer his patient to a centre equipped with the necessary scales.

8. In the circumstances now prevailing, if a baby requires milk from the Relief Board, the nurses have to spend much time in signing orders. This is a waste of time, and altogether the whole system, as it operates at the moment, is not a satisfactory one.

One can see at once that a network of centres, working in the way I have outlined, scattered as they are throughout the country, must exert an enormous influence on the education of mothers and so on the health of babies. It is but necessary to look back a few years in order to realize the great change in public opinion that has taken place.

It is now an easy matter to get mothers to weigh their babies regularly, to do all in their power to keep the baby on the breast, and to abandon night feeding. Gone are the two-hourly feed and the popularity of patent foods. There is now a sort of

public conscience making itself felt, and the mothers are prepared to learn their "A B C" in infant management. The work of the centres and the interest taken in infant welfare of late years have led to the establishment of some clear-cut rules and simple measures which, when adhered to, lead to the health and happiness of the infant, not to mention that of the family in general.

When one thinks of the chaos ruling in this department of medicine not so very many years ago, one can see a very great advancement towards a better state of affairs.

The work of a welfare scheme such as this in South Australia is attended with many difficulties of a kind not easily dealt with. Necessarily the work of the nurses brings them into close contact with the work of the medical practitioner, and in the past there has at times been friction between the two professions—the nurse on the one hand and the medical practitioner on the other.

Definite rules, copies of which are supplied for your perusal, have been laid down for the guidance of nurses, and an effort is made to see that they are kept, but, of course, difficulties arise.

Recently an advisory board has been formed to direct the medical policy and to adjudicate in troubles that occur, and medical practitioners are asked to report to the board any case in which they think the nurse has been at fault.

This board consists of representatives from various important institutions concerned with maternal and child welfare. The personnel is as follows:

Queen's Home Representatives: Dr. T. G. Wilson, Dr. E. Britten Jones.
Adelaide Children's Hospital Representative: Dr. R. Thorold Grant.
Adelaide Hospital Representative: Dr. F. Beare (on leave).
Mothers' and Babies' Health Association Representative: Dr. Marie Brown.
Mareeba Babies' Hospital Representative: Dr. F. N. Le Messurier.
Chairman: Chief Honorary Medical Officer of Mothers' and Babies' Health Association.

One must admit at the outset that some nurses are unwise, that some medical practitioners lay themselves open to criticism, and that patients, in this instance the mothers of the babies, are unintentionally mischief-making.

The complaints one hears are: (i) The nurses have changed a food ordered by a medical practitioner. (ii) They have advised a change of medical attendant. (iii) They have recommended a specialist. (iv) They have treated a sick baby.

As medical practitioners and as individualists, painful as the fact may be, we have to realize that these nurses, from constantly observing babies and doing one type of work, may know more along certain lines than we do ourselves. It must be difficult indeed for a nurse who has become expert, to stand by and watch a medical practitioner slowly starving a baby, omitting to examine the ears, or failing to diagnose a pyloric stenosis; yet these things have occurred.

I do not for a moment suggest that the nurse should interfere in any unorthodox way, but at times the temptation to do so must be great. She is told, when in doubt, to refer the mother back to her own medical attendant, and if necessary to visit the medical attendant and place the facts of the case before him. This is difficult in actual practice, the medical practitioners are busy and so are the nurses; as a profession we are loath to take advice even from the seniors in our own profession, and to accept a suggestion from a nurse is altogether beneath our dignity. It would, however, be often to our own advantage if we encouraged the nurses to report their difficulties to us direct, as misunderstandings might be avoided; and as in some cases the nurses have access to information not available to us, fresh light might be thrown on conditions otherwise obscure.

The nurses are not allowed to change the food ordered by a medical practitioner, but if a change seems desirable, the mother is referred back to her medical attendant. Very often what actually happens is that the mother goes to the nurse without telling her that a doctor has ordered the food, and even may deny that the baby is under the care of a medical practitioner; if she makes some change, the mother causes mischief on her next visit to the practitioner by saying that not she, but the nurse, is responsible. A word with the nurse concerned would be of real value.

The nurse sometimes is asked to recommend a medical practitioner to a new arrival in a district. So far instructions given are that the names of medical practitioners resident in that area should be supplied. When asked to recommend a specialist, they are not given any power to do so. The complaint is sometimes heard that the welfare nurses take away work from the medical practitioners, and in so far as their work is truly preventive, this is so; but much that they do we should find irksome and time-absorbing. Which of us, for example, wants to carry out a series of test feeds? We must realize also that the nurse, far from removing work from the medical practitioner, often puts it in his way by referring to him mothers who, if left alone, would probably consult the local pharmacist.

The problem of the child welfare nurse is the problem of the ancillary services in general, the pharmacist, the dentist, the masseuse and, nearer home, the radiologist and the various specialists. Cooperation in all these departments is to be aimed at, otherwise there is confusion and waste of energy.

Vague accusations and retaliations are not helpful; they lead only to ill feeling. Cases in which the medical practitioner has fault to find with the welfare nurse should be reported in detail to the chief medical officer, and steps will be taken to investigate the matter. This has been done in some cases with advantage to all concerned.

The fact that there are difficulties and imperfections in the working of such a scheme in the community should not be an excuse for condemning a very admirable system, a very admirable body of

workers. Each of us can do something to bring about better cooperation in this work, so that the energies of all concerned may be concentrated on the attainment of still greater efficiency.

Child welfare work is most truly preventive medicine. By right feeding, management and guidance the nation to be is profoundly influenced. The end is of a value apparent to all. The need is for wider diffusion of sound knowledge in the profession, for greater utilization of the means at our hand and for greater cooperation in the adapting of such organizations to meet the varying needs of communities and individuals.

In such schemes the efficiency of the medical profession is all-important. By it stands or falls the whole edifice. We are the leaders, and our knowledge, enthusiasm and capacity for cooperation influence profoundly the workers in all other departments. The eyes of all look to us for guidance and we must not fail them.

SUDDEN AND UNCERTIFIED DEATHS.[1]

By STRATFORD SHELDON, M.B., Ch.M. (Sydney),
Sydney.

OUR Secretary requested me a little while ago to father a paper under the title "Sudden Death". On consideration, I thought it better to enlarge the ambit by including uncertified deaths. Some years ago, in conjunction with my esteemed and learned colleague, Dr. A. A. Palmer (we have been associated for twenty-eight years), I gave you an account of our experiences in *post mortem* examinations. On that occasion we endeavoured to lay before you human conduct, emotions and passions in their unbridled and primitive form as they unfolded themselves to us. That was a subject of absorbing interest, as bearing on the practice of our branch of medicine rather than on medicine itself.

Tonight I am confining myself more or less to the medical aspect, and any opinions offered must be understood to be personal, though much of the material has been derived from our common stock.

Death is inevitable, philosophy *per se* cannot tell us why this must be. We have to look to the study of living matter and its attributes. Death is not inherent in living matter, the unicellular organism does not die. Multicellularity connotes division of labour, and the corollary is that when this division of labour is permanently upset, death occurs. In germ cells death is not inherent; but it is inherent in somatic cells because of their functional differentiation. Somatic death is the cessation of function of the three vital organs, followed by the signs of disintegration and decomposition of the tissues in general. It is not necessarily accompanied by all the component parts, for we know how tissues can be kept alive *in vitro* when parted from the body.

However, there is a trinity of organs, circulatory, respiratory and nervous, each of which is indispensable, and any one of which, sufficiently injured, brings about somatic death. There are other organs almost equally essential, and though they may not cause such rapid death, their removal brings inevitable death—adrenals, parathyreoids and pituitary.

Rarely is seen the natural process of death—the quiet sleep of old age. There is usually a terminal infection; in fact, terminal infection is the usual finale in progressive disease, hence the aphorism of Osler: "The individual rarely dies of the disease from which he suffers, save when it is an acute infection."

Avoiding all casuistry, cessation of either circulation, respiratory interchange or action of the nervous system brings about cessation of the action of the other two, and signs of disintegration follow.

The margin for a resurrection is extremely brief. When is a man dead?

Recently I was summoned up a few floors in a building to see a collapsed man laid out on a table; he was cyanosed, flaccid and motionless. I said he was dead. After a minute or so he made a convulsive attempt at respiration, and at another good interval, another. It was quite five minutes from the time he ceased ordinary breathing, collapsed, and I was sent for, until the time he made this last convulsive effort. I will leave it to you to say when he was dead.

Since January, 1898, we have been keeping an index of the causes of death in the Sydney morgue, and the principal ones are:

Heart disease	143
Arteriosclerosis	77
Aneurysms	23
Fractured skulls	144
Head injuries—	
Without fracture	11
And other injuries	120
Septicæmia (nearly all after abortion)	133
Status lymphaticus	11
Cerebral hæmorrhage	56
Drowning	70

In 1902 there were 320 inquests and in 282 cases inquests were dispensed with. In 1930 the figures were 749 inquests and in 521 cases inquests dispensed with. It is rather remarkable how close the average of inquests keeps under different headings. At random I took the years shown in the following table:

Cause of Death.	Year.					
	1915	1916	1920	1921	1924	1925
Poisoning	47	34	57	54	52	53
Drowned	41	50	68	54	38	53
Electrocution	1	0	2	3	4	6
Cut Throat	21	11	18	20	19	17
Anæsthetic	13	2	11	11	17	19
Hanging	16	8	18	18	9	15
Abortions	11	18	24	30	31	44

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 26, 1931.

In a great many cases of sudden and other deaths reported to the coroner, the history appears to be so clear that opinions are given without *post mortem* examinations. For instance, a rose pink cadaver with the head in the gas stove, hangings, a man found dead and burned by electric high tension wires, many cases of people seen to fall into the water, and bodies recovered without marks of violence, and many others, look as if the cause of death is obvious.

However, it is well to be very cautious, and the more one's experience, the less positive one becomes. I have no doubt that there are cases that have got past us. A ready story and the terminal paragraph in the police report which states there are no suspicious circumstances, all help to put one off his guard.

Three examples will help to impress this need for caution.

A young Japanese is found, dressed in his bathing suit, washed up in the sand on the beach while others are bathing. As his immediate previous movements were unknown, I did a *post mortem* examination, and on opening the body, I immediately got the characteristic smell of cyanide. Further inquiries revealed he had been crossed in his love affairs.

A woman was found in a house, hanging to the bedpost, with a buggy rein tied round her neck, by her daughter, who had been to the "Zoo" for the day. Following investigations by the police, and her false teeth being found, broken, away from the body, a *post mortem* examination was made. It was found she had injuries quite apart from the hanging. An interesting sequel to this case was that the husband, a builder and house repairer, was tried, found guilty, and appealed; the Supreme Court upset the verdict; he was rearrested; the Crown appealed to the High Court, who altered the Supreme Court's finding and ordered a new trial. He was tried again. In the second trial there was a builder on the jury who asked to see the rein by which she was suspended. He examined the knot; it was definitely a builder's knot, and no woman would have tied it. The man was convicted and got a life sentence. Lesson: do not interfere with knots or anything else about the body.

A few months ago Dr. Palmer had a case in which a patient had been in hospital three or four days, being taken there unconscious and certified as suffering from a cerebral hæmorrhage or uræmia. For some reason he did a *post mortem* examination and found a bullet in the brain, which had been fired through the hard palate. On inquiry, the people in the house knew quite well the man had suicided, but said nothing.

The Circulatory System.

Now let us examine some of the causes of sudden death. I will start with the circulatory system.

Heart Disease.

The commonest cause of sudden death, apart from injury, is heart disease, and of heart diseases, coronary disease and its consequences are by far the most frequent. Claude Bernard, in speaking of the causes of death, said:

The anatomic viewpoint is quite insufficient, and the changes which are demonstrated in the cadavers after death, give characteristics for the recognition and classification of disease rather than lesions capable of explaining death.

The application of this dictum is that anatomical changes, though clear cut, are only the substratum on which the tragic drama of the passing of life plays itself; these are no different just after death than they were minutes, hours, days, perhaps months prior to the sudden happening. So these findings can be added to whatever other information is available in the determination of the cause of death. Environmental circumstances often are illuminating in determining the cause. This is where experience is of great advantage. Twenty-seven years ago, in the case of my own father, who died suddenly, I signed the report as heart disease; now I know quite well he had a pontine hæmorrhage.

The actual cause of death, in the case of any man receiving less than £750 a year, in employment by another, has become of acute interest to the family of the deceased since the passing of the *Workers' Compensation Act*. The contentions and theories put forward to associate their employment with the cause of death do credit to the ingenuity of their medical and legal advisors, and really excite one's admiration.

You all know of the House of Lords decision in the case of "Glover, Clayton and Hughes".

A man was tightening a nut, his foot was seen to slip and he fell back dead. *Post mortem* examination showed a rupture of a large aneurysm of the aorta, and medical evidence was that death might have happened from any slight exertion or without any exertion, as in a natural act. Death was held due to an accident.

Following this decision, it is obvious that immense possibilities are opened up to associate a man's work with his death.

Returning to our coronary disease. Years before the introduction of the electrocardiograph I was satisfied from histories given and *post mortem* examinations made, that the symptom known as *angina pectoris* was associated with coronary disease, and have so often found occlusions or partial occlusions, ruptures and embolisms, and local degenerations from occlusions as to leave me without doubt as to cause and effect. Now calcareous and contracted atheromatous, partially obliterated coronary arteries, found *post mortem*, have existed for some time prior to death, and presumably there must be some determining factor which upsets the balance. The last straw, not infrequently actual clotting, is found in the atheromatous vessel. However, the actual determining factor is often not obvious. If an embolus is of such a size as to cause sudden death, I think it would be obvious, notwithstanding much expert opinion to the contrary which has been given recently in the courts.

What has struck me as remarkable is that frequently one will find one vessel diseased and almost obliterated and the other healthy, or again the first part of the vessel will be healthy and the lumen clear and the distal parts of the same vessel will be diseased. Coronary obstruction, too, is seen with healthy coronary vessels, but the opening from the aorta is obstructed by calcareous matter. Local degeneration from an occluded vessel accounts for most cases of ruptured ventricle, whether or not preceded by a local aneurysm of the vessel. The

coronaries are not truly end arteries—they do anastomose, and of late it has been stated that this anastomosis increases with each decade of life.

Valvular disease of the heart is not such a common cause of sudden and uncertified death as one would expect, but aortic disease claims a good number, and, of course, ruptured aneurysm. A dissecting aneurysm is more frequent than rupture of a fusiform aneurysm, and frequently arises from ulceration of the intima and media without any actual swelling of the aorta.

A recent case from K—. is of interest.

A young man was much grieved at the death that day of his sister, who was known to be suffering from valvular disease. He complained to his remaining sister at night of not feeling well. He died suddenly early next morning. He had early pneumonia on both sides, and a remarkable heart: the right auricle was engorged with blood and distended, and the right ventricle occupied the whole of the anterior aspect of the heart. The mitral valve was very contracted and like a shirt button-hole, evidently a congenital condition. His history was that he had never been ill or complained of any distress.

Other cases of ruptured aorta are consequent on ulceration of the œsophagus, whether secondary to impacted foreign bodies or actual disease of the œsophagus. In these cases I have seen on more than one occasion blood down the whole length of the intestinal tract to the caecum.

Air Embolism.

Another cause of circulatory death might well be considered here, namely, air embolism. Experiments have been made on dogs of injecting air into their veins, and apparently has not killed them. The cases that have come before us have all been associated with the pregnant uterus.

In one case a woman's uterus was being curetted and a douche can was used to flush out the uterus, the decidua had all been removed, but the three months fœtus was still in the uterus.

Another young woman, pregnant, went to an unregistered practitioner, who used a Higginson's syringe in treating her. She died at once. He at once called for skilled assistance in the building. This occurred at 1 p.m. By 3 p.m. I was notified, and with the late Dr. Sydney Jamieson a *post mortem* examination was made at 4 p.m. Every care was taken to avoid any confusion and complication, and immediately the front of the chest was removed, the pericardium was opened and the heart examined. The right ventricle was distended, and on incision air escaped and the blood was seen to be in a frothy condition.

A few months ago a pregnant woman was in a house with her family; the others left at about 2 p.m.; she was found by a child dead on the floor of the bedroom, with a Higginson's syringe and a chamber with some Condy's fluid in it. Dr. Palmer and myself did a *post mortem* examination at 4 p.m. The same precautions were adopted, and the heart at once examined before the rest of the body was touched. On this occasion there was a distinct swish as the air escaped from the ventricle. The distended and balloon-shaped heart was characteristic. As in the former case, the *vena cava* was stripped and bubbles of of air could be seen and pushed along the vessel.

It was also noted in this case that expression of blood from the right side of the uterus produced gas bubbles, while the left did not do so. The separation of the placental area with a little hæmorrhage was distinct on the right side.

I related this case to one of our best known pathologists. He asked whether we attempted to get cultivations from the blood of the heart and veins. I told him we did not. He said he was not convinced; he was left cold.

In 1910, lacking experience, I did a *post mortem* examination on a young woman whose body was found, fully clothed, except for her drawers, in the sand behind a racecourse. She had a considerable amount of blood coming from the os; the amnion was not torn. The bleeding was traced to a separation of the placenta.

I found nothing else in the body to account for death. A nurse was tried for causing this woman's death—she was actually seen from the other side of the street doing something to her. Owing to insufficient evidence she was acquitted.

There are a number of other similar cases. There is no doubt in my mind that the use of a syringe or douching tube from which the air has not been expelled, accounts for these deaths. There is always a separation of the placenta, and the amnion remains intact, and invariably the heart is found distended, with frothy blood exuding, and usually air bubbles can be distinctly seen and pushed along the *vena cava*.

Cerebral Hæmorrhage.

Another cause of death from interference with the circulation is cerebral hæmorrhage. Perhaps it would be more fit to consider death as resulting from interference with the nervous system, because, although it is due to a ruptured vessel, death is really the result of pressure on the nerve centres. The most common situation is rupture into the posterior part of the cerebral hemisphere. I do not propose to traverse the subject—exhaustive accounts may be seen in any text book on medicine—but to offer a few observations. Some years ago I published a series of cases of cerebral hæmorrhage in quite young women; these all occurred in the anterior lobe. In only one case was a cause suggested, as the woman suffered from a chronic leuchæmia.

The condition of one of these women had been diagnosed as hysteria, and she had been under treatment as an out-patient at hospital for over twelve months.

A month ago there was a young policeman, twenty-five years old, who had been diagnosed as suffering from *encephalitis lethargica* five years ago, but had been quite well since and passed into the service after a very strict examination. He complained of headache during the night and died very rapidly in the morning. He had a large hæmorrhage in his anterior lobe; all his vessels seemed healthy and there was no sign of disease elsewhere.

Another remarkable case was that of a young married woman who was confined of her first child at 6 a.m., had a cup of tea at 8 a.m., and died almost at once. She had a typical pontine hæmorrhage. She had never been ill in her life, and had a normal confinement and apparently was a perfectly healthy, fresh young woman.

I have no doubt, if a *post mortem* examination had not been done in this case, the condition would have been described as a pulmonary embolus. What is the determining factor in a cerebral hæmorrhage? I am frequently asked in court as to the influence of a man's work. My experience is that the rupture takes place without any reference to actual exertion. More hæmorrhages occur when a man is at rest,

sitting down or in bed, and I cannot call to mind a case in which hæmorrhage occurred when a man was actually straining at some particular work.

Of course, it would be foolish to deny the possibility of an extra raised blood pressure due to strenuous exertion being the determining factor and last straw. This is a very different thing to a man being engaged in his ordinary avocation, say, shovelling earth or wheeling a truck. In doing a *post mortem* examination one frequently, on opening the abdomen, gets a shrewd idea that it is a case of cerebral hæmorrhage. An over-distended bladder shows that the cerebrum has been out of action for some time before death.

The other day, in response to my colleague's exclamation of "finished already", I was vaunting my perspicacity, but I found he knew all I did when I saw a full bladder. Most deaths from cerebral tumours have been diagnosed or tentatively diagnosed before death.

Meningitis.

Frequently the presence of meningitis has not been suspected before death. In suppurative meningitis the mode of extension of the infection is usually obvious, whether it be from the middle ear, nose or fracture of the skull; at times, however, it is very obscure.

Status Lymphaticus.

The next mode of death which I wish to bring before you, is the condition known as *status lymphaticus*. *Status lymphaticus* has been described as a hyperplasia of the lymphoid tissue, shown by enlargement of the pharyngeal, thoracic and abdominal glands, as well as enlargement of the spleen and thyreoid, with a persistent thymus. Confining ourselves to the adult cases since 1928, very briefly, the cases are as follows. They are Dr. Palmer's cases.

A jockey, aged eighteen years, started to dress himself and fell back with a gurgling noise, dead. *Post mortem* a persistent enlarged thymus, a spleen twice the normal size, and many enlarged mesenteric glands were found. The heart was distended.

A female, aged seventeen years, died soon after a tonsil operation (Kelem). The thyreoid and thymus were much enlarged, the spleen weighed 360 grammes (twelve ounces), the mesenteric glands were enlarged. The legs were big and pitting, there was no œdema. She was a big girl and weighed 88·2 kilograms (fourteen stone).

A boy, playing cricket in the roadway, sat down suddenly and died. The lungs were voluminous and distended, the right heart was distended. The thymus weighed over 60 grammes (two ounces); section revealed thick chocolate coloured fluid. Cervical, mediastinal, mesenteric, inguinal and axillary glands were enlarged.

A female, aged twenty-one years, an out-patient at hospitals, complained of something in her chest and died as a medical practitioner arrived. The thymus weighed 52·5 grammes (one and three-quarter ounces). On section the thymus was normal, but had hæmorrhages through it. The spleen weighed 375 grammes (twelve and a half ounces). A lingual tonsil was found.

A female, aged twenty-two years, felt "queer", lay on her mother's bed and died at once. The thymus weighed 22·5 grammes (three-quarters of an ounce), the spleen 225 grammes (seven and a half ounces). Analysis revealed no abnormality.

A girl, aged fifteen, was playing a simple game and collapsed. The thymus was large and the spleen twice the normal size. Some petechial hæmorrhages were present on the heart.

A.H., aged thirty years, a woman, was staying with people and returning to her husband in a motor car. She started to vomit and was very ill. She was put into the car again and taken to a doctor and died on the way. The thymus was large and the spleen large; analysis revealed no abnormality.

There are two more I have already published.

V.C.W., aged twenty-nine years, had been swimming on a Saturday afternoon; he felt ill when he came out and took a bus to a doctor at North Sydney. He collapsed in the waiting room and died almost at once. *Post mortem* he was an athletic man, the only abnormality was a persistent thymus, weighing 0·88 gramme. The spleen weighed 22·5 grammes. The heart muscle was excellent. Nothing abnormal was found in the brain, including the hypophysis.

F.G., aged thirty-eight, was moving a heavy printing press with a lever; he collapsed, lay down for a few minutes and died on the way to hospital. The thymus weighed 50 grammes, the spleen was twice the normal size. This last was the subject of a workers' compensation claim. I expressed the opinion that death was contributed to by strenuous exertion. The claim was disallowed; the defence suggested some minute cerebral hæmorrhage undiscovered by me.

I have no explanation to offer as to what the condition is or how it affects the bodily economy. I have read articles in which the whole thing is treated as a myth, but these *post mortem* findings and their practically universal absence in other cases leaves me with an unshaken belief in the condition of *status lymphaticus* as a definite cause of death.

In the literature there are a good many cases quoted of death before operation in which no anæsthetic had been administered, and there were *post mortem* findings similar to those just described. I am offering no theory or explanation of how or why these people die, but merely wish to state the cases and indicate a promising field for investigation for some of our younger members.

Suffocation.

Under the heading of suffocation I should like to direct your attention to the extreme suddenness of death. I have had at least nine cases of men, usually elderly men, dying suddenly in an eating house, and finding, *post mortem*, their glottis blocked with a lump of meat. It is very remarkable the large size of the lumps of meat that these people attempt to swallow or get caught on the top of their larynx.

The usual history is that they are at the table and fall back dead without a suggestion that it is due to choking.

Some years ago a young woman, aged nineteen years, had been at the Coast Hospital suffering from diphtheria. After one month's convalescence she had several swabbings taken and declared free from infection. Being a few days at home, she was sitting at her meal, collapsed and died. *Post mortem* it was found that five centimetres (two inches) of a cast of her trachea had become separated, jambed into her glottis and choked her.

Of course, without a *post mortem* examination this woman's condition would have been classed as a post-diphtheritic vagus neuritis.

If I have not exhausted your patience, I should like to bring to your notice two or three other causes of death.

Poisonings.

What impresses itself on me is the extraordinary change which has taken place with regard to the poisons causing death in the last twenty-eight years. When first I did the work, phosphorus was in common use; children used to eat match-heads, and disappointed young women used to swallow them. At that time, "rough on rats" was also in vogue. This was followed by regular epidemics of lysol poisoning. I have seen as many as four bodies in the morgue at one time, killed by lysol. There is no question that the cessation of the publishing of names of the poisons has diminished the incidence. Carbon monoxide poisoning still holds its own, and cyanide has come much into vogue. Strychnine has always been popular and is still a frequent means used.

I am distinctly of opinion that far fewer forlorn young women poison themselves than formerly.

The newer synthetic narcotics are now used to a great extent, and are frequent causes of death, either accidental or by design.

Death from Abdominal Lesions.

Treves long ago talked of peritonism, meaning by that, shock from disturbance of the peritoneum as a rapid cause of death.

A boy at midnight complained of pain in his abdomen for the first time and sent for a doctor at 7 a.m., but was dead before the doctor arrived. An incarcerated small intestine due to a band attached to the end of a Meckel's diverticulum was found.

An attendant at the Town Hall, at work till midnight without complaint, died at breakfast time. A strangulated hernia was found *post mortem*.

Neither of these persons had time to develop peritonitis or get poisoned from obstruction.

I have seen several cases of ruptured duodenal ulcer in which death has taken place very rapidly, long before any possibility of the results of septic absorption.

Other Conditions.

I have not time to go into the subject of death following criminal abortions. The unbelievable and extraordinary injuries which have been found after abortions attempted at the fourth month would take too long to recount.

We have had a number of cases of a fatal issue after attempted abortions in which the woman has had amenorrhœa, either apart from pregnancy (I have had two in which there was acute miliary tuberculosis), hydatidiform mole, or in which there has been an extrauterine pregnancy unrecognized and has ruptured.

Deaths following criminal abortion still average nearly one a week, and this can only be a very small fraction of the enormous number which are done and are not followed by death. I saw it stated that at the new arbortoria in Russia there are 40,000 abortions carried out every year.

Deaths after abortions are of three kinds: (i) Rapid septicæmia, (ii) purulent peritonitis, not so rapid, or peritonitis and septicæmia combined, (iii) chronic cases or pyæmia with fungating growths on the cardiac valves and septic infarcts in the spleen, lungs, kidneys and, less seldom, elsewhere.

I have made practically no reference to mistakes and delinquencies in diagnosis and treatment. These, of course, do occur, but anything gross is extremely rare, and it would ill become me to parade them here.

Unjustifiable and careless death certificates, given from the best of motives, may bring trouble on the donors, especially when a patient has not been recently attended. Things are not always as they seem, even with X rays.

A man, operated upon and successfully treated for a growth in his bladder, a year later developed chest symptoms. He was examined with X rays and clinically, and it was settled that he had malignant recurrence in his lung. One day he fell off a tram as he was mounting and fractured his skull. *Post mortem*, one side of his chest was full of purulent fluid and his collapsed lung was like shrunken leather up against his spinal column. I understand the physician still believes in his diagnosis, notwithstanding my finding.

Conclusion.

I thank you for your patience, and if the personal pronoun has occurred too frequently, forgive me, because, as I told you in the beginning, these are personal views on personal experience, and nobody could be more conscious of their shortcomings than I. I could go on with much more. I have not touched on injuries or deaths by violence and will now make room for my colleague, who is going to discourse to you on bullet wounds.

In conclusion, I trust you each and all will have a sudden if not an uncertifiable death.

ANTE PARTUM HÆMORRHAGE.[1]

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ANTE PARTUM hæmorrhage, as a complication of pregnancy or early labour, is a condition of great importance; it is at times one of the gravest complications by which the obstetrician may be confronted, especially if met with under conditions and in surroundings that make correct treatment difficult or impossible.

Ante partum hæmorrhage may be due to one of two conditions, namely, hæmorrhage due to the separation of a normally situated placenta and called accidental hæmorrhage; or hæmorrhage from a placenta abnormally situated in some portion of the lower uterine segment (that is, *placenta prævia*) and designated unavoidable hæmorrhage, for bleed-

[1] Read at a meeting of the Section of Obstetrics and Gynæcology of the New South Wales Branch of the British Medical Association on March 18, 1931.

ing must occur during the later months of pregnancy or early in labour.

ACCIDENTAL HÆMORRHAGE.

Considering accidental hæmorrhage first, it is difficult to estimate correctly the percentage number of such cases, as undoubtedly many occur in which, even though the hæmorrhage be more than slight, the patients do not present themselves for examination or treatment. We have all had the experience of seeing definite evidence of previous hæmorrhage on examining a placenta at the time of a patient's confinement. Among the last 2,386 confinements at the Royal North Shore Hospital of Sydney, only four cases of accidental hæmorrhage have occurred requiring treatment, and all four patients were *multiparæ*, in whom such hæmorrhage more frequently occurs. It is of the utmost importance to determine to which of the two conditions mentioned the hæmorrhage is due. The ability or inability to force the presenting part into the pelvis gives useful information, as in *placenta prævia*, especially if the placenta is situated close to the os, it will be found that the presenting part will not become engaged. But accurate diagnosis necessitates a careful but thorough examination *per vaginam*, if necessary under an anæsthetic, when the examining finger must be passed carefully through the os and the lower uterine segment palpated. If no placenta can be felt, then one is almost certainly dealing with a case of accidental hæmorrhage.

Ætiology.

Various theories have been put forward as to the cause of this condition. Paramore[1] attempts to prove that raised blood pressure is the cause and that the rupture of the placental sinuses is produced mechanically. He claims that the factors in play are the general (that is, the aortic) blood pressure, the pressure of the blood in the placental sinuses and the pressure of the *liqtor amnii*, and that when accidental hæmorrhage occurs in toxæmic conditions it is not the toxæmia which causes the hæmorrhage, but the raised blood pressure. It is difficult to accept this theory; it is certainly not in accord with clinical facts. Another theory put forward is that accidental hæmorrhage is due to a previously existing infective endometritis. Certainly the condition is more ilkely to occur in *multiparæ*, some of whom might be presumed to have suffered from an infection of the endometrium; but this theory does not help to prove any causal relationship. Moreover, according to Shaw,[2] chronic endometritis is a comparatively rare disease. Syphilis and trauma have both been brought forward as causative factors; but, of course, many cases occur without any history or evidence of either playing a part.

The theory most generally accepted is that it is due to a toxæmia based on the presence of a previously existing chronic nephritis, and there is most interesting experimental evidence in support of this view.

In *The Journal of Obstetrics and Gynæcology of the British Empire* F. J. Browne and Gladys Dodds,[3] of University College Hospital, give results of various experiments on rabbits. To quote from their report:

> The rabbit is a peculiarly suitable animal for the experiments mentioned, because the time of mating and period of pregnancy are known accurately. Intravenous injections can be easily given and, finally, the placenta in its essential structure is similar to that of the human subject. In a series of non-pregnant rabbits a chronic oxalate nephritis was induced by the administration over a period of about three months of a 1% solution of sodium oxalate at frequent intervals. The rabbits were then mated, and on the twentieth day of pregnancy (a period corresponding to the sixth month in the human subject) a fresh injection of sodium oxalate was administered, in order to set up an acute exacerbation of the nephritis. When this had occurred, an emulsion of *Bacillus pyocyaneus* was injected intravenously, and in every case external accidental hæmorrhage occurred shortly afterwards. In some cases there was also concealed retroperitoneal hæmorrhage, with placental infarction and extravasation of blood into the walls of the uterus and subsequent separation of the muscle fibres.
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> In other experiments, instead of the sodium oxalate on the twentieth day, uranium nitrate, which causes an acute nephritic condition, was injected with the same result. This shows that sodium oxalate on the twentieth day is not the cause of the hæmorrhage, but from further experiments they showed that, provided chronic nephritis is present, there is no need to inject uranium nitrate on the twentieth day of pregnancy. Hæmorrhage can be caused by the injection of *Bacillus pyocyaneus* alone; but, further still, if chronic nephritis is present, then the injection of uranium nitrate on the twentieth day will cause hæmorrhage without the subsequent injection of *Bacillus pyocyaneus*. In the absence of chronic nephritis, the injection of *Bacillus pyocyaneus* is not usually sufficient to cause *ante partum* hæmorrhage, even though the organisms set up an acute nephritis. In none of ten experiments carried out to test this point did bleeding occur.
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> In the absence of chronic nephritis the injection of uranium nitrate, so as to cause an acute nephritis, is not usually sufficient to cause an *ante partum* hæmorrhage. It occurred in only one experiment out of ten and placental infarction occurred in another. In three animals suffering from known chronic nephritis spontaneous *ante partum* hæmorrhage occurred in the second half of pregnancy. In one it occurred twice, and in another four times in successive pregnancies. In those animals with chronic nephritis albumin may be absent from the urine between pregnancies and during the early part of the pregnancy itself, but in the latter half of pregnancy and before the onset of spontaneous hæmorrhage albumin appears in the urine. This seems to have an important bearing on the question of the so-called recurrent toxæmia of pregnancy. The conclusion is that *ante partum* hæmorrhage is not due to the organisms, but probably to the failure of the kidney to excrete poisons, which accumulate in the circulation, and ultimately, when in sufficient concentration, lead to the hæmorrhage. An alternative explanation is that the kidney has normally a metabolic function, which is interfered with in chronic nephritis. All the evidence from these experiments goes to show that the one important point predisposing to the *ante partum* hæmorrhage is the presence of a chronic nephritis.

I have quoted these results rather extensively as they appear in the article referred to, since I consider them to be both of great interest and extreme importance.

If these conclusions are correct, and the experiments certainly give very strong evidence in support, then the question arises: should a woman who has had a severe accidental hæmorrhage be pre-

vented from going through a subsequent pregnancy, not merely on account of the danger of another hæmorrhage, but because of the risk of further damage being done to kidneys already in a state of chronic nephritis?

Symptoms.

The symptoms vary greatly in severity. In many cases they are slight. In other cases of revealed hæmorrhage, after the initial hæmorrhage, which often ceases, no further bleeding or other symptoms arise. In others, of course, the bleeding is much more severe. As in most instances there is concealed as well as revealed hæmorrhage, the gravity of the condition and the severity of the symptoms depend largely on the amount of concealed hæmorrhage. When hæmorrhage is totally concealed, the patient exhibits all the indications of severe loss of blood and also of shock, for in such cases there is no doubt that shock plays a very important part. In addition to the general symptoms there is rapid onset of severe abdominal pain, the uterus is hard, swollen and tender, the fœtal parts cannot be felt nor the fœtal heart sounds heard. In these circumstances the fœtus is almost invariably dead, on account of the more or less complete separation of the placenta from the uterine walls. The worst condition of all is that described as utero-placental apoplexy, when the blood percolates through the uterine walls, even along the tube and to the peritoneal cavity. Such hæmorrhages as these are due to the degeneration of the uterine muscle, which is unable to withstand the pressure of blood within the uterus. The condition of the patient is desperate and demands prompt treatment. She is pale, with rapid, feeble pulse, subnormal temperature, cold, clammy skin and sighing respiration; in fact, there is the typical appearance of a patient *in extremis* from both hæmorrhage and shock.

Treatment.

In many cases no treatment is required beyond rest, quiet, a light diet, and perhaps a dose of morphine, as either the symptoms are slight or the hæmorrhage has ceased early. The patient should be kept at rest in bed for at least a week after all hæmorrhage has ceased. In the presence of severe external bleeding more active treatment is required. In a case of external accidental hæmorrhage, with the patient not in labour, should the membranes be ruptured? I find many different opinions on this point from equally eminent obstetricians. Berkeley and Bonney[4] do not favour this course. According to Bourne,[5] in "Recent Advances in Obstetrics and Gynæcology", Hastings Tweedy strongly advocates it. Edgar[6] is rather in favour of rupturing the membrane, whilst Johnstone,[7] of Edinburgh, is opposed to this method of treatment, and the text book in use at the Queen Charlotte Home is in favour of it. For my own part, if the bleeding is external and the patient not in labour, and facilities and conditions generally are suitable for packing the vagina, I prefer to adopt this method of treatment without rupturing the membrane. Rupturing the membrane requires an anæsthetic and must be thoroughly done with sterilized wool plugs wrung out of an antiseptic (preferably flavine, one in a thousand). The bladder must first be emptied and then every six hours whilst the plugging remains, as there is certain to be difficulty in passing urine. Dry wool swabs are useless, as they shrink on becoming moist. In addition, a dose of morphine may be given and a tight abdominal binder applied, fixed to a vulval pan. Labour almost always commences in a few hours. If not, the plugging should not remain *in situ* for more than twelve hours, but should be removed. Probably no further bleeding will occur. If it does, further plugging may be carried out after carefully cleansing the vagina. If the patient comes into labour and no further bleeding takes place, the case may then be treated as one of ordinary labour without undue interference. If, however, bleeding does occur when the patient is in labour, the membranes may be ruptured; in a vertex presentation a de Ribes bag may perhaps be introduced, and in a breech presentation a foot may be brought down, any malpresentation being first corrected if necessary. An injection of pituitrin and the application of a tight abdominal binder will be of material assistance. On the expulsion of the bag delivery may be either left to Nature or forceps applied to the head. If necessary in a breech presentation, delivery may be hastened by gentle traction. If facilities or conditions are not suitable for plugging, then membranes may be ruptured; but I prefer, if possible, the patient to come into labour before this course is adopted.

In cases of severe concealed accidental hæmorrhage where the uterus is hard and swollen and tender, if the patient's general condition is fairly good and proper facilities are available, then Cæsarean section may be considered; but probably the best course is to give a full dose of morphine and pituitrin with perfect rest and quiet. The objective is to get the uterine muscle to recover its tone and ability to contract, which will be indicated by the bleeding becoming revealed externally. When this occurs, then, the question of Cæsarean section may be considered, or the case may be treated, as before mentioned, as one of external accidental hæmorrhage. In utero-placental apoplexy, where the severity of the condition is indicated by the continued and progressive symptoms, Cæsarean section must be performed. Many advocate in addition immediate hysterectomy; this appears to me to be a desperate treatment in such an extreme condition. I think I would prefer a section, tying the tubes (a matter of only a few seconds) and I should try by every means, such as pituitrin, ergot, hot towels *et cetera*, to improve the uterine condition. If no facilities for operative treatment are available, then the only course to adopt is the giving of morphine, injection of pituitrin, application of an abdominal binder and, of course, all means must be used to combat the loss of blood and the shock by warmth, hot bottles, blankets, saline infusion, transfusion *et cetera*.

UNAVOIDABLE HÆMORRHAGE.

In unavoidable hæmorrhage or hæmorrhage from a *placenta prævia*, the placenta varies in its situation as regards the undilated internal os. The symptoms are external bleeding of varying severity, usually after the seventh month of pregnancy or early in labour, and the diagnosis is made by feeling the placenta *per vaginam* as mentioned beforehand.

I know of no theory brought forward which gives a satisfactory reason for the condition. Whether the position of the uterus at the time the fertilized ovum gets into the uterus has any bearing on the condition is so far a matter of surmise. This type of hæmorrhage is of rather more common occurrence than accidental hæmorrhage. Among the last 2,386 cases of labour at the Royal North Shore Hospital of Sydney, there were treated 21 cases of *placenta prævia*, with one death—a case to which I shall refer later, as it teaches a useful lesson.

Treatment.

Under suitable conditions, with a live child of a viable age, I feel sure that the correct treatment of central *placenta prævia* is Cæsarean section. This gives a satisfactory result for both mother and child. I would also advocate the same treatment in a marginal *placenta prævia*, particularly if a living child is desired by the parents. Failing suitable conditions for operation, if the patient is not in labour, the vagina should be plugged with the same precaution, of course, as previously mentioned. The membranes should not be ruptured if the patient is not in labour. At the onset of labour and when labour pains are definitely established, the plug should be removed and two fingers should be passed into the uterus to one side of the placenta if possible, otherwise through the placental tissue itself, bipolar version being performed if necessary. Then a foot should be brought down through the os; this will effectually check the hæmorrhage; a weight may be applied to the foot if thought advisable. In some cases it may be possible to perform external version prior to the intrauterine manipulations; this is a method to be advocated.

I remember, when practising in the country many years ago, being called on one occasion about twenty-five miles into the bush, the message being that the patient was pregnant, but bleeding badly. On my arrival the hæmorrhage had fortunately ceased and the patient's condition was fairly good, but a central *placenta prævia* was felt. With every possible care the vagina was plugged under an anæsthetic, administered by the patient's own mother, under instruction, who had to take frequent jaunts to the door or window for fresh air. After some hours the patient came into labour, and when the pains were well established, the plug was removed. Severe bleeding started afresh. My knowledge of abdominal palpation was at any rate less than it is now, and I was immensely pleased when passing my fingers through the os and placental tissue, to feel a foot which I was able to seize and pull through the os. The child was still-born, but the result was perfectly satisfactory as regards the mother.

In performing Cæsarean section for *placenta prævia* there is one point against which the operator must be on his guard. Although the uterus may be firmly contracted and everything progressing satisfactorily, the patient's condition may rapidly become worse, owing to hæmorrhage from the placental site, which does not tend to contract, the blood escaping between the patient's thighs on to the table. The condition may become serious before this is recognized, unless the operator is on his guard, the symptoms possibly being attributed to shock.

In a marginal *placenta prævia*, when the patient is not in labour, the same treatment may be adopted. If the patient is in labour and section has been decided against, then probably the best treatment is to bring down a foot after version, if this is necessary. A de Ribes bag may be inserted if the patient is in labour and the os is sufficiently dilated; but in a low-lying marginal *placenta prævia*, on the expulsion of the bag it may still be necessary to bring down a foot to check the hæmorrhage that is likely to recur. The drawback to the bringing down of a foot is the high percentage of fœtal mortality; perhaps 50% at least of the fœtuses are still-born. But it is certainly a most effective method of checking the hæmorrhage. In a lateral *placenta prævia* the symptoms will probably not be so urgent, at least in the early stages. In such cases, when the patient is in labour and the lie of the fœtus a longitudinal one, the rupture of the membranes may be quite sufficient to check the hæmorrhage, especially if the presentation be vertex. If necessary, a de Ribes bag can be inserted and extraction can be made with forceps when dilatation has occurred; or, in breech presentation, a foot may be brought down.

These cases of *placenta prævia* always cause anxiety, for in addition to the initial hæmorrhage, which may be severe, there is the risk of infection and *post partum* hæmorrhage. A fair proportion of cases require manual removal of the placenta on account of hæmorrhage, and it is the trickling of blood subsequently which may turn the scale against the patient unless she be carefully watched. On account of the danger, scrupulous care should be taken to prevent the risk of sepsis. I know men even now who boast they do not wear gloves in their maternity work; this, to me, seems quite wrong. In the event of sepsis occurring in a case treated by such a practitioner, and if it were proved that he did not wear gloves, would his position be perfectly secure from a medico-legal point of view?

Once the *placenta prævia* is definitely diagnosed, plans for treatment should immediately be adopted. The only circumstances in which delay is admitted are if the patient is under hospital conditions, when treatment can be initiated at once if necessary. Yet even in a hospital one may meet with trouble.

Some months ago a patient was admitted to the Royal North Shore Hospital of Sydney with a central *placenta prævia*; bleeding had ceased, and as I was due in the theatre twenty-four hours later, I decided to wait, for the convenience of all concerned. That morning at 2 a.m. I had to hasten to the hospital on account of a further severe hæmorrhage. Cæsarean section was carried out, fortunately with satisfactory results for both mother and child.

It will no doubt be noticed that there is one method of treatment I have not touched upon, and that is traction on the infant's scalp by forceps, such as Willett's forceps. I have had practically

no experience of this treatment and hope to hear the results and opinions of members who may have adopted it. The method appeals to me and, I think, would be particularly useful in cases of vertex presentation, when the placenta was not situated too low in the lower uterine segment. Whatever method is adopted, no intrauterine manipulations should be attempted until all necessary appliances are ready to hand. The intrauterine manipulations are almost certain to cause further bleeding, hence the necessity for all apparatus and appliances to be ready and available. With the exception of Cæsarean section, it will be recognized that all methods of treatment are calculated to promote slow delivery; to attempt to bring about rapid delivery of a patient with *placenta prævia* who is suffering from the effects of hæmorrhage, is courting disaster. Here may I refer to the one death before mentioned, at the Royal North Shore Hospital.

The patient was sent into hospital from a neighbouring suburb over five years ago, with a low marginal placenta. On arrival, she was obviously suffering from the effects of hæmorrhage, the os was well dilated, with a vertex presentation. Attempts were made to effect delivery with forceps; these failed and the patient's condition became urgent. Version was performed and a foot brought down. Hæmorrhage was thus checked, but by this time the patient's condition was desperate; she did not rally, and the tragedy of a woman dying undelivered had to be faced.

Two mistakes were made in attempting forceps delivery. First, the rule that delivery from a patient suffering from the effects of hæmorrhage must not be hastened was violated. Secondly, the placenta, acting as a wedge below the head and preventing it from engaging, really made the operation a high forceps operation. The placenta also altered the line of axis of the application of the forceps, although the os was well dilated. Immediate version, with drawing down of a foot, might have given a better result; it would certainly have been a better mode of treatment.

In any discussion that may take place on the merits or demerits of my remarks this evening, I trust that members will not confine themselves to consideration of measures that should or should not be adopted in the conditions under which we carry out our work, with first-class facilities almost invariably to hand. They should remember also the man in the country, possibly called out into the bush, working single-handed, with a bad light, his only assistant being perhaps a frantic mother or mother-in-law, whose knowledge of asepsis is a menace rather than a help. I have had that experience, and I know what it means.

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AN INVESTIGATION INTO THE RELATION OF ACHLORHYDRIA TO CARCINOMA OF THE STOMACH.

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DURING the years 1924-1928, inclusive, 10% of all deaths in Australia were caused by cancer, while in 3·5% the cancer originated in the stomach. During this time, in 66% of all deaths, the age was forty or over. As all but 5% of cancers occurred in people of forty or over, this means that about 15% of the deaths of all people of forty or more were due to cancer, and 5% to cancer of the stomach. The last figure is the same as that quoted by Hurst(1) for the percentage of deaths from gastric cancer in people over forty in England. Early diagnosis, the only hope of successful treatment, is only too rare. Anything that can throw new light on the ætiology of this disease may lead to results of great value.

In this investigation we have taken as our starting point the common association of gastric cancer with achlorhydria or hypochlorhydria. This association has received many explanations in past years, in all of which, however, it was assumed that the loss of acid secretion was the result of the cancerous growth. It occurred to us that the achylia or hypochlorhydria might precede and indeed be a factor in the production of the growth. This was first suggested by our finding of achylia in two normal healthy young people, one parent of each of whom had died of gastric cancer, and as it has been shown by various observers (Hurst,(2) Apperly and Norris(3) and others) that achylia commonly runs in families, there occurred to us the possibility that the affected parents likewise had a constitutional achylia, preceding their carcinomata.

Hurst(1) records two cases of carcinoma of the stomach in both of which achlorhydria had been found three years before symptoms of the growth appeared. Porges(4) records several cases in which achlorhydria was found preceding by many years the symptoms of carcinoma. Hurst(1) also quotes three cases of cancer appearing in the stomachs of patients suffering from pernicious anæmia, a condition almost always preceded by achylia.

The achlorhydria preceding malignant change might of course be secondary to some condition such as chronic gastritis, or be a congenital constitutional condition. We decided to investigate the latter possibility.

Since our work began in 1926, however, Hurst(1) has published a most interesting and important paper also showing that achlorhydria precedes cancer of the stomach, but his evidence seems to indicate that the achlorhydria is due partly, at any rate, to chronic gastritis. Our work is therefore the complement to his investigations. Part of his conclusions are as follows:

1. In about 75% of cases the carcinoma is secondary to chronic atrophic gastritis; these comprise a large majority of those

with a short history, almost all of those with achlorhydria, and most of those with hypochlorhydria.

2. In about 20% of cases the carcinoma is secondary to chronic gastric ulcer: these comprise a large majority of those with a long history, all of those with hyperchlorhydria and a high normal curve of acidity, and almost all of those with the average normal acidity.

3. In most of the remainder the carcinoma is secondary to a simple adenoma of the stomach.

To decide if congenital achylia precedes most cancers of the stomach is obviously a very difficult task, and results obtained by almost any method of investigation can be regarded as suggestive only.

Our method of attack is an indirect one, and depends upon the important fact that gastric types or diatheses, including achylia, run in families. This is very clearly shown in some recent work by us.[3] We investigated, by means of the fractional method of gastric analysis, gastric function in thirty-one families, comprising eighty-six individuals, and found that (*a*) in ten families (twenty-nine individuals) there was a close similarity in all respects between all members of each family; (*b*) in eleven families (twenty-nine individuals) the similarity was less complete; (*c*) in six families (twenty individuals) there was no similarity between members of the same family, or the resemblances were too few for classification under (*a*) or (*b*); (*d*) in four instances when parent and child were examined, in three both parent and child gave the same results.

The argument on which our investigation is based is as follows:

(*a*) Achylia is a common condition often appearing in the several members of the same family. If one parent in each of a series of families was known to have true constitutional achylia, we would expect roughly half the children of the series also to have achylia. Similarly, we might expect a condition of achylia in a large proportion of the brothers and sisters of a series of people with congenital achylia.

(*b*) Conversely, if about half of the children and brothers and sisters of a series of patients with carcinoma of the stomach have achylia or a low hypochlorhydria, we might expect that a high proportion at least of the patients themselves had the same condition as a congenital defect, long before they developed carcinoma.

In order to test these suggestions we examined, by means of the fractional method of gastric analysis, one hundred blood relatives (sons, daughters, brothers and sisters) of people who had been proved, by operation or by *post mortem* examination, to have suffered fron carcinoma of the stomach. Only those relatives who were free from gastric symptoms were examined. The investigation extended over a period of more than four years.

Results.

At first it seemed that our results would indicate that achylia was common as a congenital condition among carcinoma subjects, for the group of relatives showed a proportion of people with achylia (21%) and hypochlorhydria (21%) four or five times greater than that found among normal students (Bennett and Ryle[5], Apperly and Semmens[6] and others).

Table I shows a comparison of our figures with those of these authors. It is, however, well known

TABLE I.

Classification, by their Gastric Secretory Curves, of (a) a Number of Relatives of Patients with Carcinoma of the Stomach, and (b) for comparison, a Number of Healthy Young Men (by various Observers). (Figures given as percentages.)

Observers.	Number of Cases.	A Achlorhydria.	B Hypochlorhydria	C Low Normal.	D Normal.	E High Normal.	F Hyperchlorhydria.	Columns C, D, E Combined.
Bennett and Ryle[5] ..	100	4	1	10	59	18	8	87
Baird, Campbell, Hern[6]	57	2	7	26	40	14	11	80
Apperly and Semmens[6]	90	8	4·5	5·5	42	19	21	67
Above groups combined	247	5	4	12	49	17	13	78
This series:								
All Cases	100	21	21	13	23	11	11	47
Sons	41	7	12	17	27	17	20	61
Daughters	36	28	28	11	25	3	6	39
Brothers and Sisters	23	35	26	9	13	13	4	35

to those familiar with test meal work that females on the whole have a lower acidity and a higher proportion of achlorhydria than males, and old people than young. It became obvious in fact that we could not fairly compare our group of mixed relatives, many of them elderly, with the young male student groups of Bennett and Ryle (Figure I) and of other

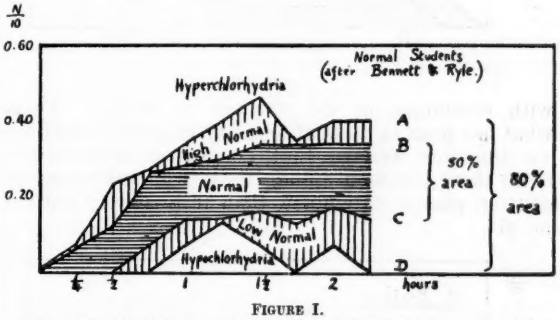

FIGURE I.

When Bennett and Ryle examined 100 normal students by means of the fractional test-meal it was found that 50% of the curves fell between the limits B and C and 80% between the limits A and D. The remainder were above or below these limits.

workers. For a more accurate comparison, therefore, we divided our relatives into four groups, namely, sons, daughters, brothers and sisters of patients with carcinoma of the stomach. The results are shown in Figures II, III, IV and V. It will be seen from

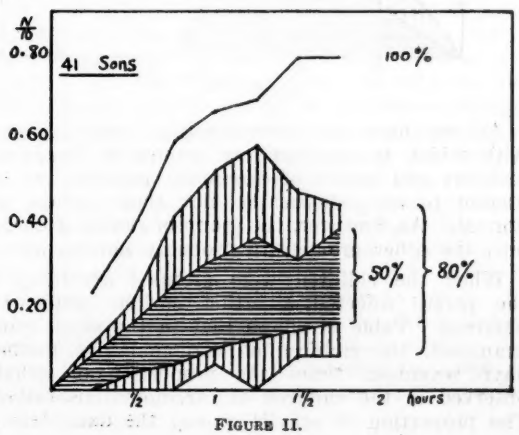

FIGURE II.

these Figures and from Table I that the sons, nearly all young men, have nearly the same gastric acidity as the student groups. Unfortunately, nobody, so far as we are aware, has made graphs of normal young women, older women and older men with which to compare Figures III, IV and V. As, however, there is such a very close resemblance between Figures I and II and between the figures for sons and for the student groups (especially the Australian student group) in Table I, we can definitely state that gastric function among the sons of patients

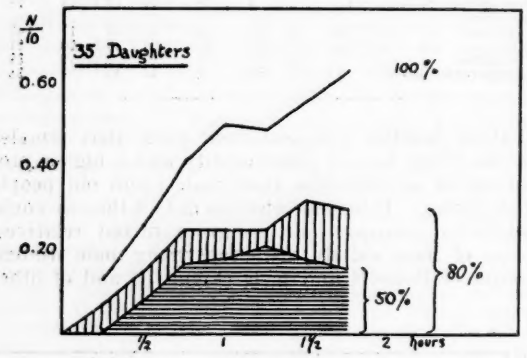

FIGURE III.

with carcinoma of the stomach is normal. From what has been said about heredity in gastric function, we therefore believe that congenital achlorhydria is no more common among those who subsequently develop gastric carcinoma than it is among normal people.

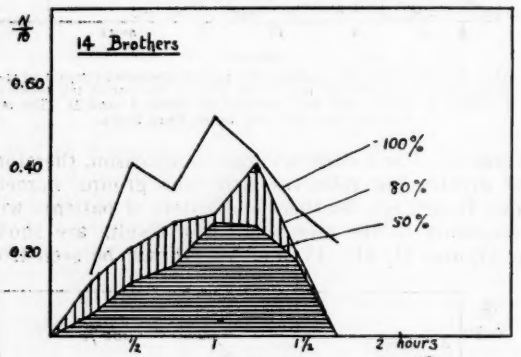

FIGURE IV.

As we have no corresponding normal groups with which to compare our groups of daughters, brothers and sisters of carcinoma patients, we are unable to say whether or not these groups are normal. As, however, the group of sons is a normal one, the other groups are probably normal also.

When the children were grouped according to the parent affected, a rather curious result was obtained. Table II shows that in the small group examined, the children of carcinomatous mothers have seventeen times the proportion of achylia observed in the children of carcinomatous fathers. The proportion of achylia among the daughters of the former group (56%) is extraordinarily high. Furthermore, in only one individual of this group was the free acidity above 20 (0·020 N.) whereas in the latter group high acid curves were fairly

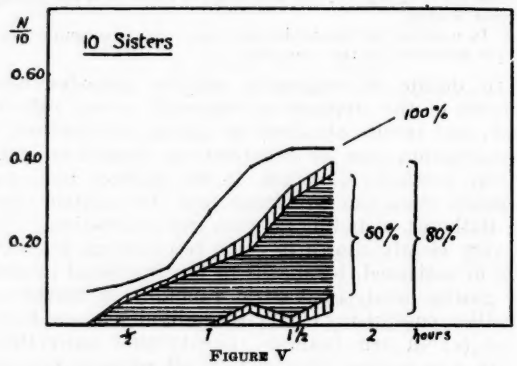

FIGURE V

common (45%). Between the groups of sons there was very little difference. Whatever significance these observations have, if any, it is difficult to say.

TABLE II.

Showing the Incidence of Achlorhydria among the Children of Patients suffering from Carcinoma of the Stomach.

Groups.	Numbers.	Number with Achylia.	Percentage.
Sons of Carcinomatous Fathers ..	25	0	0
Daughters of Carcinomatous Fathers	20	1	5 } 2·2%
Sons of Carcinomatous Mothers ..	16	3	19
Daughters of Carcinomatous Mothers	16	9	56 } 37·5%

Among our subjects of cancer age (over thirty-five) were seventeen men of whom seven had achylia (41%) and fourteen women of whom six had achylia (43%). In one hundred healthy old people examined by Dedichen[7] achylia was found in 80% of the men, and in 60% of the women.

A comparison of the student groups with the above middle-aged and elderly groups shows the increase of achylia with age.

Conclusions.

There is much evidence that the achlorhydria that so commonly accompanies carcinoma of the stomach really precedes that condition. Achlorhydria may be acquired, the result of chronic gastritis, or it may be a congenital familial peculiarity. Our work, by indicating that the achlorhydria is not of the latter variety, supports Hurst's contention that it is the result of a chronic gastritis preceding the appearance of carcinoma.

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(4) O. Porges: "Magenkrankheiten", 1929, pages 163 and 164 (quoted by A. F. Hurst in reference 1).
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NASAL SINUSITIS IN RELATION TO LOBAR PNEUMONIA.

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ONE hundred and three *post mortem* examinations of the nasal accessory sinuses, constitute the basis of this paper. The cases were not selected in any way; they were carried out on patients who had died in the wards of the Melbourne Hospital and who came to *post mortem* examination in the ordinary routine way. The period of time elapsing after death till the time of *post mortem* examination, varied from a few hours up to twenty-four hours. In very few cases had a record been made of any examination of the nasal sinuses, carried out during the final illness. The diagnosis of the condition of the sinuses was formed from their macroscopical appearance.

The method of examination was for Mr. Rogers, the *post mortem* assistant, to remove the calvarium and take out the brain. I then examined the base of the skull, particularly those structures in near relation to the sinuses. The bony roof of the sphenoidal sinuses was then removed and the lining membrane carefully examined. The contents and dimensions of the sinuses were also noted. The bone forming the roof of the anterior and posterior ethmoid cells was removed on each side, these cells being thus opened from above. The cranial wall of the frontal sinuses was then removed and these cavities inspected. The maxillary antra were entered through an incision along the superior margin of the upper gums and the anterior bony walls were removed, the contents of these sinuses being thus thoroughly exposed.

A frontal mirror and electric light were used to examine the interior of the sinuses as they were opened up.

In eleven of these one hundred and three cases, the *post mortem* diagnosis of lobar pneumonia was made by Dr. Wright Smith. In every one of these eleven cases acute infection of one or more of the nasal accessory sinuses (100%) was present. In ten cases the anterior sinuses (maxillary antrum, anterior ethmoids, and frontal sinuses) were acutely infected. In half (five) of these the posterior sinuses (sphenoidal and posterior ethmoidal cells) were also infected. In one case only was a posterior sinus alone acutely infected.

We may now consider the affected sinuses in an endeavour to determine whether one particular cavity is more prone to be inflamed than another in association with lobar pneumonia.

We divide the cases into the following three groups:

In Group I sinuses of the anterior group (maxillary antra, anterior ethmoids or frontal sinuses) were acutely inflamed.

In Group II sinuses of both the anterior (maxillary antra, anterior ethmoids or frontal sinuses) and the posterior group (sphenoidal sinus and posterior ethmoid cells) were acutely inflamed.

In Group III sinuses of the posterior group (sphenoidal sinuses and posterior ethmoid cells) were alone acutely inflamed.

Group I.

Group I cases are shown in the following tabulation. In this summary the word infected signifies that the lining mucosa of the sinus showed definite signs of inflammation. "N.A.D." signifies that nothing abnormal was detected, that is, no macroscopical signs of inflammation were found. "R." signifies right, and "L." left.

Case XXXII. Male, aged 49 years. Autopsy October 7, 1930.
Maxillary Antrum: R. infected membrane. L. Pus, acute suppuration.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Anterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Posterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Sphenoids: R. N.A.D. L. N.A D.

Case XL. Female, aged 30 years. Autopsy November 2, 1930.
Maxillary Antrum: R. full of pus, acute suppuration. L. full of pus, acute suppuration.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Anterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Posterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Sphenoids: R. N.A.D. L. N.A.D.

Case LV. Male, aged 53 years. Autopsy January 15, 1931.
Maxillary Antrum: R. no actual pus, thickened œdematous membrane, acute inflammation. L. N.A.D.
Anterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Posterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Sphenoids: R. N.A.D. L. N.A.D.

Case XCV. Male, aged 58 years. Autopsy April 9, 1931.
Maxillary Antrum: R. N.A.D. L. filled with pus, acute suppuration.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Anterior Ethmoid Cells: R. N.A.D. L. Congested mucosa.
Posterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Sphenoids: R. N.A.D. L. N.A.D.

Case XCIX. Male, aged 75 years. Autopsy April 18, 1931.
Maxillary Antrum: R. membrane thickened and infected, no pus found. L. N.A.D.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Anterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Posterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Sphenoids: R. N.A.D. L. N.A.D.

From this table one finds that in three of the five cases pus was found to be present in large quantity in the maxillary antrum. In one case this purulent collection was present on both sides. In the other two cases, though no pus was found in the antrum at the time of examination, the mucosa was acutely inflamed.

The fact that the frontal sinuses are placed in an anatomical position which lends itself to better drainage tends to explain why in no case was any purulent collection found in them. In only one case (XXXII) was some congestion of the mucosa present.

The anterior ethmoid cells are also found to have their ostia placed so that their drainage according to gravity is more adequat ly provided for, than is that of the maxillary antra, and these cells (anterior ethmoid) also failed to show any purulent collection. In only one instance (Case XCV) did the mucosa of these cells show signs of congestion.

Of course one would hardly expect to find macroscopical signs *post mortem*, when the inflammatory reaction of the mucosa had been only slight. But had any cell been actually discharging pus, signs of this more marked inflammation would surely have been present.

The indications are that the principal source of infection was in the maxillary antrum. The infection of the antrum in old people is usually of long standing. The hyperplastic changes of the mucosa indicate the chronicity of the infection. The final attack is an acute exacerbation, with the formation of pus in many instances.

Pus formation is indicative of the resisting powers of the patient to the acute exacerbation of the micro-organisms' toxicity. These microorganisms have been shown to be present in the sinus mucosa (Pickworth).

In a debilitated patient actual pus may not be found *post mortem* in an acutely infected sinus, the discharge in these circumstances being a thin brownish fluid. This fluid is infinitely more irritating to the mucosa of the nose, throat, larynx, trachea, bronchi, *et cetera* than is the ordinary yellow pus. It causes intense inflammatory reaction of the lymphoid tissue of the naso-pharynx and lateral pharyngeal bands with which it comes into contact.

The general signs of an acute toxæmia are very pronounced during the presence and liberation of this thin fluid. As the powers of resistance of the patient are established, pus is formed and is found instead of this thin brownish fluid. With the appearance of pus, the symptoms of general toxæmia become less severe. Later mucopus takes the place of actual pus as the patient overcomes the acute infection.

Group II.

The cases in Group II are set out in the following tabulation.

GROUP II.—CASES SHOWING ACUTE INFECTION OF ANTERIOR AND POSTERIOR SINUSES.

Case XVII. Male, aged 41 years. Autopsy August 6, 1930.
Maxillary Antrum: R. pus and polypi present, acute exacerbation on chronic infection. L. pus and polypi present, acute exacerbation on chronic infection.
Anterior Ethmoid Cells: R. no pus, acute inflammation. L. no pus, acute inflammation.
Frontal Sinuses: R. pus in the anterior pocket. L. pus in the anterior pocket.
Posterior Ethmoid Cells: R. pus in the cells. L. pus in the cells.
Sphenoids: R. pus in the sinuses. L. pus in the sinuses.

Case XXXIX. Male, aged 44 years. Autopsy October 29, 1930.
Maxillary Antrum: R. acute infection. L. acute infection.
Anterior Ethmoid Cells: R. acute infection. L. quieter (some infection).
Frontal Sinuses: R. infected. L. quiet.
Posterior Ethmoid Cells: R. infected. L. infected.
Sphenoids: R. pus. L. pus.

Case XXXIV. Male, aged 62 years. Autopsy October 9, 1930.
Maxillary Antrum: R. pus, acute exacerbation. L. not acutely infected.
Anterior Ethmoid Cells: R. infected. L. infected.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Post Ethmoid Cells: R. pus acute. L. infected.
Sphenoids: R. pus, acute exacerbation. L. absent.
Pus similar to that in the right antrum was found on the vocal cords.

Case LXX. Male, aged 22 years. Autopsy March 1, 1931.
Maxillary Antrum: R. pus acute. L. pus acute, suppuration.
Anterior Ethmoid Cells: R. quiet. L. congested mucosa.
Frontal Sinuses: R. N.A.D. L. infected.
Posterior Ethmoid Cells: R. pus acute. L. N.A.D.
Sphenoids: R. acutely infected. L. N.A.D.

Case XXX. Male, aged 41 years. Autopsy October 3, 1930.
Maxillary Antrum: R. N.A.D. L. pus acute.
Anterior Ethmoid Cells: R. N.A.D. L. infected, no apparent pus.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Posterior Ethmoid Cells: R. pus acute. L. pus acute.
Sphenoids: R. N.A.D. L. N.A.D.

The Anterior Nasal Sinuses.

In all of these five cases there was suppuration of the maxillary antrum. In two cases both sides were affected, and in three only one.

Though the anterior ethmoids may have shown associated inflammatory signs, there was no actual pus in the cells. The frontal sinuses also may show some associated inflammatory congestion of the mucosa, but in only one case (XVII) was any pus found in the sinus, and this was in a small pocket on the floor, at the entrance of the naso-frontal duct. The lining mucosa did not appear acutely inflamed. It was noticed that it was the frontal sinus and the anterior ethmoid on the same side of the nose as the suppurating antrum that showed any inflammatory changes.

The Posterior Nasal Sinuses.

In four of the five cases pus was present in the sphenoidal sinuses; in two cases in both right and left cavities, and in two cases in the cavity on one side only.

In one case (XXX) the sphenoidal sinus showed no inflammatory changes, but here pus in marked amount was present in both right and left posterior ethmoidal cells.

In fact we see that the posterior ethmoidal cells, on one or both sides of the nose, showed acute inflammation in every case.

Group III.

The case in Group III is set out in the following tabulation.

GROUP III.—INFLAMMATION OF THE POSTERIOR SINUS ALONE.

Case XX. Female, aged 38 years. Autopsy August 18, 1930.
Maxillary Antrum: R. N.A.D. L. N.A.D.
Anterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Posterior Ethmoid Cells: R. N.A.D. L. N.A.D.
Frontal Sinuses: R. N.A.D. L. N.A.D.
Sphenoids: R. chronic infection, acute exacerbation, thin brownish fluid. L. chronic infection, acute exacerbation, thin brownish fluid.

Discussion.

It would appear after reviewing these cases that there is a definite association between the acute infection of the maxillary antrum and lobar pneumonia and that the inflammatory reaction of the lung may be caused in many cases by the entrance into the lung *viâ* the larynx of pathological organisms and their toxins that have developed in the maxillary antrum.

Amongst the conclusions that one might arrive at, are the following:

1. In all cases of lobar pneumonia, the maxillary antra should be investigated, and this is most satisfactorily done by suction and disinfection of their contents. The earlier this is done the better will be the prognosis when the antra are infected. It is remarkable the improvement in general symptoms and signs that may occur after once these cavities are treated in this way. Also it is noted how often a single "wash out" seems sufficient to clear the suppurative condition of the sinus. At the first suction pure pus may be obtained and on repeating this procedure ten days later the contents may appear free of pus.
2. In regard to pneumonia in association with influenza, this procedure is particularly advocated.
3. When cerebral symptoms arise in the course of lobar pneumonia the sphenoidal sinuses should be similarly dealt with. The suction and disinfection of the sinuses can be done with the minimal disturbance to the patient. He does not require to be

moved in his bed. General anæsthesia is not necessary; a local anæsthetic is all that is required.

The method of suction exploration of the sinuses is that described by P. Watson Williams.[1]

Acknowledgement.

I should like to express my gratitude to Dr. Wright Smith, Pathologist to the Melbourne Hospital, for the courtesy that he showed to me whilst I was examining these cases *post mortem*.

Reference.

[1] P. Watson Williams: "Chronic Nasal Sinusitis", 1930.

DRIVER'S THIGH.

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THE object of this communication is to draw attention to a condition fairly generally recognized by both medical and non-medical motor drivers and to suggest the name "driver's thigh", which seems both convenient and descriptive. If a label has already been affixed, it has escaped the writer's notice.

The affection is a neuralgia or a neuritis of the sciatic nerve observed in those who spend a fairly large part of their time driving a motor car.

Symptoms vary from an ill-defined feeling of fatigue of the thigh muscles to those of a classical sciatica.

The cause is a long continued pressure on the sciatic nerve just before its division in the lower third of the thigh. The pressure is produced by the use of the accelerator pedal, which requires the foot to be held in the one position, often for long periods (Figure I).

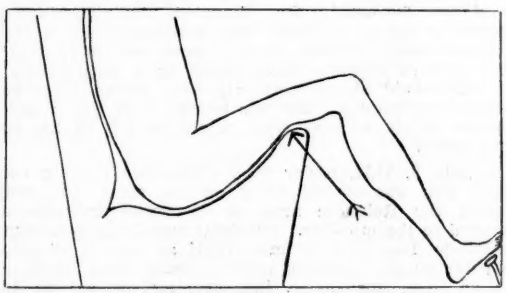

FIGURE I.
Springs in seat have given, the seat has sunk and the driver has slumped forward. Pressure occurs at the spot marked by the arrow.

Treatment is, of course, by having the seat adjusted. It would seem that the seat without the usual tilt is perhaps the best kind for the driver. The trouble comes only when the driver sits badly or the springs of the seat have given way (Figure I). Figure II shows the seat and thigh in correct relation.

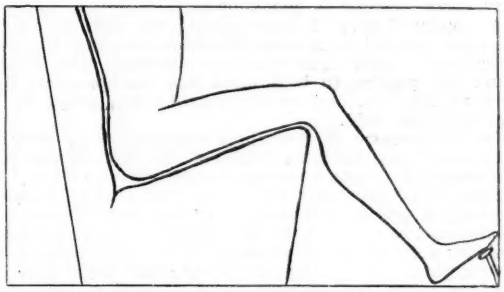

FIGURE II.
Correct position; seat has not sunk.

Reports of Cases.

PARASAGITTAL MENINGIOMA.

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Case I.

THE patient, Mrs. S., a *nullipara*, aged forty-six years, was referred to me by Dr. E. Britten Jones for admission to the Adelaide Hospital. She enjoyed good health until six years ago, when she began to get "turns", which she described as a numb feeling in the right side of the abdomen, accompanied by a desire for immediate defæcation. She just had time to perform this act, when it would be followed by a feeling of numbness and tingling, beginning in the right big toe and then slowly spreading to the right foot, leg, trunk, arm and face. These turns were experienced occasionally during the next four years, but two years ago she noticed that they were associated with twitching of the right foot (and sometimes of the right arm also), with loss of consciousness.

During the last six months the fits had become less severe, without loss of consciousness; but she began to lose power in the arm and leg. For three months her vision had been failing and she had suffered from severe frontal headaches. She was still menstruating regularly, but had put on over 9·0 kilograms (twenty pounds) in weight in six months. As far as the history was concerned, the diagnosis appeared obvious, as a tumour involving the paracentral lobule on the left side. But the chief interest in this case lay in the fact that it illustrates how the late symptoms due to actual pressure on and deformity of brain substance, apart from internal hydrocephalus, may obscure the initial symptoms and hinder accurate localization.

On admission to hospital in November, 1930, a most striking feature was very advanced mental impairment and loss of memory, so that most of her history had to be obtained from her relatives. She had alternate periods of depression and delusion. She was able to stand upright, but could not walk, as she had little control of the movements of her right leg. There were no objective sensory changes. There was marked loss of power in the right arm and leg, with slight wasting; tendon reflexes were variable, but were never more active than on the left side, and generally were absent; the right knee jerk was often pendular, the right plantar reflex always flexor. There was no spasticity. During the period of investigation a

weakness of the lower right facial muscles became obvious during emotion.

At no time was there any measurable papillœdema; there was a varying degree of blurring of the nasal sides of both disks. The visual fields were difficult to elicit, but showed very early bitemporal hemianopsia.

A varying degree of astereognosis was always present; the same applied to muscle incoordination, and the daily alteration in both signs was very obvious. The Wassermann test applied to both blood and cerebro-spinal fluid gave no reaction, and no abnormality was found in the cerebro-spinal fluid.

The radiograms showed three changes: first, irregular bony areas on the inner table of the left frontal bone and enlarged venous channels in the anterior fossa, consistent with the existence of a frontal lobe tumour; secondly, a regular enlargement of the *sella turcica*, with erosion and anteversion of the posterior clinoid processes, probably entirely due to pressure of the brain from above; and thirdly, a stippling of the parietal bone near the mid-line in the post-Rolandic area, such as is sometimes seen overlying a meningioma. A ventriculogram was made to settle the localization, as the patient's signs on admission were as much those of a frontal lesion extending backwards as of a Rolandic tumour. The left ventricle could not be tapped; it was subsequently shown to be almost entirely collapsed, but the right was tapped and found to contain only about fifteen cubic centimetres of normal fluid. After replacement with air, the radiograms showed a marked shift of the mid-line to the right, a very small left ventricle, and both ventricles displaced down towards the base of the brain. The anterior horn of the left ventricle filled with air.

A two-stage operation was performed, as profuse bleeding from the superior longitudinal sinus was encountered before the dura was opened, necessitating double ligature of the sinus at the first operation. At the second stage the tumour was entirely removed and proved to be a meningioma arising from the *falx cerebri* and the under-surface of the sinus; the tumour was egg-shaped, measuring six by three centimetres. At both operations the anæsthetic consisted of preliminary injection of morphine and hyoscine, then "Novocain" supplemented by just enough ether to keep the patient asleep.

For two days the patient was almost hemiplegic; but from then onwards she has made steady improvement.

The tumour, which was growing from the under-surface of the superior longitudinal sinus on the left side, in the angle formed between that structure and the *falx cerebri*, was definitely encapsuled and was compressing and displacing the paracentral lobule and the adjacent portion of the medial surface of the parietal lobe. This compressed brain tissue was very œdematous, almost liquefied, and subsequent events have shown that its function was destroyed. It was oval-shaped and measured 6·4 by 3·8 centimetres; on section it was found to be largely cystic and relatively avascular for a meningioma, which it proved to be on microscopical section.

The patient's condition on October 8, 1931, was noted as follows: The most favourable result is in her mental condition; admittedly this is not quite normal, as she is still subject to attacks of depression and weeping during the menstrual periods. This tendency to weep easily seems to be very common in patients whose brains have been subjected to pressure over a long period. But otherwise she takes a normal interest in her life and surroundings and does her own housework cheerfully, as far as her leg permits. She can read, but tires easily. She has recovered good movements of both arm and leg, but is still entirely astereognostic. For this reason, and because she lacks the precision of movements of the joints, her writing is poor, she can crochet, but cannot use a needle; and though she can hold a knife quite well, she lacks the precision wherewith to use it properly.

This patient had evidence, clinically and radiographically, of pituitary dysfunction before her operation. Since the relief of intracranial tension, her menstrual periods have been resumed, and she has lost about 9·0 kilograms (twenty pounds) in weight, but this is offset by an enormous appetite.

Case II.

The second patient was referred to me by Dr. H. F. Shorney and Dr. H. W. Wunderly, to whom he had been sent for the loss of sight, and to them I am indebted for permission to publish these notes.

S.F.P., a married man, aged sixty-one, gave the following history on July 5, 1931. Up till three years ago he believed himself to be in excellent health, and he had served in both the South African and European Wars (in the latter as a member of a remount unit). Then, with no preliminary illness, he had several fits in one day, with loss of consciousness; residual symptoms consisted of a feeling of stiffness of the left arm and leg. This feeling lasted some weeks and then gradually wore off. Eighteen months ago he noticed headache and occasional diplopia. One year ago he became unconscious for three days and noticed after recovery that his vision was failing. From that time he steadily lost the use of his left arm and leg, and since January, 1931, had not been able to walk at all. Since February, 1931, he has not been able to recognize his relatives. For more than six months he had lost control of his bladder; he perceived the desire for micturition only when he was actually passing urine; on rare occasions he had time to ask for a urinal. He had a good past history; since the original fits he had been treated as suffering from cerebral hæmorrhage. There had been no vomiting. In the last few months the relatives had noticed failing memory and mental dulness, but no other changes in personality. There had been no change in weight.

Neurological examination was made on July 5, 1931. The patient was about 88·2 kilograms (fourteen stone) in weight, he was completely bedridden. His mental processes were slow, he had frequent yawning and scratching of the nose. Headache was not so intense as it had been. Vision practically was nil; very rarely he could distinguish light from darkness. Moderate bilateral papillœdema was present, with very advanced bilateral secondary optic atrophy. Bilateral abducens palsy was present. There was left seventh nerve palsy of the central type. Left twelfth nerve palsy was present. There was complete loss of use of the left arm; the spasticity and tendon reflexes varied from day to day, a sign very suggestive of a cortical lesion. There was no astereognosis and no sensory changes were found. Spastic paralysis of the left leg was present in extension. In the right leg there were defective voluntary movements and increased tendon reflexes. No sensory changes were found in either leg. There was no mass reflex. Micturition occurred from sixteen to twenty-four times a day. The systolic blood pressure was 145 and the diastolic pressure 100 millimetres of mercury. X ray examination revealed little except dilated venous channels running to the great wing of the sphenoids.

The impression recorded at the time was that the patient had a tumour involving the right frontal lobe, extending back to the arm and leg motor areas, probably a meningioma or a tumour invading the cortex. The chronicity favoured the diagnosis of meningioma. Admittedly the examination was incomplete, and a really good stereoscopic X ray picture might have helped us to make a more accurate localization, as a meningioma of this size would almost certainly have shown some bony reaction overlying it. But his urgent need was a decompression to try to save what might be left of his optic nerve fibres.

On July 9, 1931, under 0·5% "Novocain", a big osteoplastic flap was turned down on the right side, which exposed the Rolandic area in its posterior angle and extended to the mid-line. The local anæsthetic was supplemented by less than 15 mils (half an ounce) of chloroform during an operation lasting nearly three hours, and was an ideal anæsthetic, just keeping the patient in a drowsy condition throughout. Difficulties were encountered from a very tough skull, with profuse bleeding from venous channels in the bone. The tumour was found to be much further forward than was anticipated, the posterior extremity of what was obviously a large meningioma showing in the extreme antero-medial angle of the exposed area. As it was highly improbable that he would have survived the blood loss inevitable in the removal of such

a tumour, the wound was closed in anticipation of a second stage operation.

This is where Cushing's technique is so invaluable. It enables one to reopen a skull flap within a few days without fear of wound or meningeal infection. As he points out, the removal of a meningioma is associated with more hæmorrhage than when any other brain tumour is concerned; and to be able to get the preliminary part of the removal done in a first stage operation means that the surgeon approaches the most delicate part of his work feeling fresh (for raising a big osteoplastic flap in a tough skull can be exhausting work), and in the intervening days the patient's blood supply can be increased by a transfusion, if the first stage has been very wet.

During the next six days the flap bulged considerably, and he became much brighter as the result of decompression. There was a little voluntary movement of the left knee and ankle joints. The wound was healed by the sixth day. Daily enemata of concentrated magnesium sulphate had reduced the bulging of the wound.

On July 17, 1931, under a similar anæsthetic, the anterior and medial limbs of the flap were reopened and a fresh incision made from the anterior angle across the mid-line towards the left outer canthus for 5·0 centimetres (two inches). The frontal bone was nibbled away forwards and across the mid-line, the longitudinal sinus and the tumour being exposed. It soon became evident that the exposed portion of the tumour was but a small part of it; and in the hope of minimizing brain trauma, an endeavour was made to lessen the size of the tumour by Cushing's method of excavating it with the endothermy loop. This is necessarily a slow procedure, and as the enormous arteries of the tumour could not be properly controlled, rapid removal *en masse* became necessary. The anæsthetist, Dr. Thorold Grant, gave the patient an intravenous injection of thirty ounces of gum saline solution, while the tumour was rapidly enucleated; it was deriving its blood supply from two big arteries in the falx, and these were tied. In the bottom of the cavity was seen the genu of the *corpus callosum*. The enucleation caused a drop in blood pressure from 130 to 85 millimetres of mercury. The wound was carefully closed, with one rubber tissue drain, and he left the theatre with a blood pressure of 100 millimetres and a pulse of 120, able to speak. Within an hour he had a rigor, probably due to the gum saline solution, but he quickly recovered.

With one exception, his progress has been remarkable. Unfortunately, that one exception is his sight, which has improved only to the extent that he can always tell light from darkness. Seventeen days after the second operation he was able to sit up in a chair and could move his left leg freely. He said his head felt clearer than it had for months, and he became bright and cheerful and interested in the daily news.

On October 3, 1931, he was in excellent general health, eating and sleeping well. He has full control of micturition. He walks without assistance and without a stick, but must, of course, be given his direction. He has almost full use of his left hand. There is no facial paralysis and no abducens palsy. The tongue still deviates a little to the left. His sister reports that his mentality is as good as ever, except that he becomes lachrymose when the question of his vision is raised.

The tumour is oval-shaped, weighing 98 grammes, and measures 8·0 by 6·5 centimetres and 4·5 centimetres in depth.

Dr. L. B. Bull reports:

> Sections show a meningioma. It appears to be well encapsuled. The tumour cell is a large flattened cell, often arranged in large syncitial-like masses. The tumour is very vascular and the tumour cells are in close association with the vessels. The vessel walls commonly undergo a hyaline change and sometimes large areas of hyaline material are to be seen apparently originating from the vessel walls.

This patient bears out the theory that a centre for the control of micturition exists in the frontal cortex. The loss of control so often seen in these frontal lobe lesions has been ascribed to mental impairment, so that the patient does not know or care if he wets his bed. This man was fully aware of the act of micturition and was acutely unhappy over his loss of control.

Reviews.

HISTOLOGY.

ON account of the rapid expansion in recent times in medical science, the tendency nowadays in many medical schools seems to be to concentrate, as much as possible, on those aspects of the basic medical sciences which find a direct application in clinical medicine. From the point of view of the production of practical medical men and dentists, this tendency represents probably the application of business methods in medical education, and there may be something to be said in its favour. There have been so many advances, too, in technical methods that the subject matter in the basic sciences has expanded enormously. This is especially so in the science of histology. We are not surprised, therefore, to see new text books appearing which represent attempts to save the student's time by presenting the most important facts in a concise manner without too detailed a discussion of theory. This apparently has been the aim of Dr. Piette in writing his new text book of histology,[1] and we think he has achieved it. The book certainly presents in a handy and easily assimilable manner the essential facts of present day histology and those likely to be required by the medical and dental student. The book contains no directions for laboratory work, but an excellent and succinct account of the essentials of histological technique is given. The section on cytology is especially well prepared, and the subject matter has been chosen carefully and wisely so that, although only about thirty-five pages have been devoted to this rapidly expanding part of the science, it is quite complete and adequate for the purpose for which this book has been written. Another section that deserves special mention is that on the digestive system, including, of course, the teeth. The other chapters call for no special criticism, but all reach a generally high standard and show a careful sifting of the facts and concise method of presentation. The illustrations are carefully selected and for the most part well reproduced. The book as a whole is well printed on good paper, and the plan is adopted of printing in bold type each technical term when it first appears. Important phrases and classical quotations are also printed in bold type to give them greater emphasis. The author should be particularly competent to write a book of this kind, as, although he is now a pathologist, he was formerly engaged in teaching histology and embryology.

THE TEETH.

THE first edition of "Those Teeth of Yours", by Dr. J. Menzies Campbell, was published in 1929. The enthusiasm with which it was received and the speedy appearance of a second edition are welcome indications of the growing realization that dental decay demands more than the daily use of a tooth brush and prompt submission to the dentist's drill if it is to be kept in check.[2] Dr. Menzies gives a brief but interesting survey of the history of dentistry followed by a simple description of the anatomy of the teeth and jaws, and then discusses "The Whys and Wherefores of Dental Caries". He insists on the supreme importance of a proper dietary containing the necessary vitamins. This, with oral hygiene and the maintenance of

[1] "Textbook of Histology for Medical and Dental Students", by E. C. Piette, M.D.; 1931. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 476, with 277 illustrations, some in colour.

[2] "Those Teeth of Yours: A Popular Guide to Better Teeth" by J. Menzies Campbell, D.D.S., L.D.S., F.R.S.E.; Second Edition; 1931. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 162, with 17 illustrations. Price, 3s. 6d. net.

good health, will largely prevent decay of the teeth. For the treatment of established decay various fillings and crowns are described. There is a comparatively lengthy chapter on *pyorrhœa alveolaris*, which, he points out, is an early symptom of scurvy and which he claims is curable, and one on "Extraction of Teeth and Insertion of Artificial Dentures". The best chapter is, however, that entitled "Children's Teeth". It is properly divided into two sections. Section I, "The Expectant and Nursing Mother and Her Responsibility". The author writes: "In other words, the diet of the expectant mother is the alpha and the omega of preventive dentistry." We agree that it is the alpha, but if it is also the omega, then Section II, "The Child", should be unnecessary, and also the rest of the book. In Section II he quotes a lengthy paragraph written in THE MEDICAL JOURNAL OF AUSTRALIA by Dr. A. Jefferis Turner, and beginning: "It would almost seem that we feed our children expressly with the object of destroying their teeth".

The chapter on "Teeth in Relation to Health" deals with the paths of infection from the teeth leading to disease in other parts of the body. There is a formidable list of diseases given in which the infection may have originated from the teeth and the part played by the absence of teeth as a cause of indigestion, the possibility of cancer arising from the trauma of a broken tooth, and the indirect effect of painful teeth on the health are indicated. There is, however, practically nothing said about apical absorption and sepsis arising from the efforts of dentists to preserve teeth which ought to be extracted. On the contrary, the author states most emphatically that pulpless teeth are not necessarily devitalized and that they should be filled and crowned. He mentions root abscess, and there is an X ray film of a tooth (not filled) showing the condition, but he appears to have steered clear of the subject as far as possible. Many medical practitioners will feel that he has not been honest in the matter. Many patients suffering from focal sepsis and the diseases attributable to it are found to have devitalized teeth which have been root filled or crowned at variable periods previously. Some of these unfortunate people have spent a considerable amount of money on the tamping of these anaerobic culture media, and so it is often difficult to convince them. It would perhaps be no exaggeration to say that, in the experience of many general practitioners, apical sepsis in filled and crowned teeth is the most common single ætiological factor in the production of disease. In comparison, pyorrhœa is distinctly rare.

MEDICINE AND LAW.

THE subject matter of "Medical Jurisprudence", by Carl Scheffel, is not such as is usually found in works with this title.[1] The author states that his intention is to show the medical practitioner how and in what ways the law affects him in his medical or surgical practice. This he does under such headings as: "The Contractual Relations of Physicians", "Physicians and Torts", "Witnesses and Evidence", "Property Interests of Physicians", *et cetera*. Part only of this information is usually provided in works on forensic medicine, part in works on ethics, and much the practitioner leaves to his legal adviser.

Although dealing with the law of the United States of America, the book contains much valuable information. In "Physicians and Torts" the author treats at length with actions for malpractice which, he states, are increasing by leaps and bounds, and ascribes, perhaps rightly, much of this to careless or malicious statements by medical practitioners themselves, and criticizes the eagerness of some medical men to take the witness stand for the purpose of testifying against their brother practitioners. Often, no doubt, this is done for the purpose of gain or advertisement, and some medical men become almost professional witnesses. His suggested remedy, however, is not likely to be adopted. It is that every member of a medical society should be forbidden to volunteer testimony in a malpractice action against any member unless and until a board of fellow practitioners authorized for that purpose by the society has reviewed the essentials of the proposed testimony to be given. One can easily imagine the outcry if the medical practitioner was refused permission to give the evidence, whilst, on the other hand, permission given by such a board or committee would load the evidence against the defendant.

The author states that in a few States in which physicians have given unfair, unjust and unscientific medical testimony in such actions, the medical examining boards have revoked their licences or rebuked them on the grounds of unprofessional conduct.

An interesting chapter deals with property interests of physicians. It is stated that a patient's ownership of a prescription ends when the chemist hands him the medicine, the ownership being now transferred to the chemist. It has been ruled by the courts that in ordinary photography the negatives remain the property of the photographer, and the Radiological Society of North America has assumed the same attitude in connexion with skiagrams. It appears, however, to be laid down by some courts that the medical man taking the X ray picture holds it in trust for the patient and is precluded from using such property for purposes which may possibly run counter to the patient's interests, the X ray examination having the status of a privileged communication. This, however, the author argues, is not absolute and does not prevent the use of it for furtherance of knowledge, provided the interests of the patient are not in any way jeopardized.

[1] "Medical Jurisprudence", by C. Scheffel, Ph.B., M.D., LL.B.; 1931. Philadelphia: P. Blakiston's Son and Company. Demy 8vo., pp. 325. Price: $2.50 net.

DARWINISM AND DISEASE.

EDMUND HUGHES has, in his little brochure, "Seasonal Variation in Man", expressed his dissatisfaction with our present static pathological conception of disease and backed up his criticism by argument and speculation revolving round a means of harmonizing the Darwinian doctrine of human descent with an understanding of the problems presented by human disease.[1]

The author first became engaged in this thesis by noting a certain seasonal behaviour in cases of rickets; and he has sought evidence in the implication of human phylogeny which may result in fresh and far-reaching biological conceptions of disease. To use his own words: "Just as the botanist would be unable to understand the physiological behaviour of a deciduous plant, except in relation with the regular changes of the physical year, so it may not become possible to understand the *raison d'être* of some of our diseases and the general plan of the organic singularities on which they depend, until they are described in the same relation."

The book is frankly philosophical in tone, and for the average clinician will prove rather difficult to read. Our impression is that the author has certainly become possessed of a pregnant idea, but that even with a wealth of anthropological examples, he has not carried it through its complete gestation. The reader is in consequence frequently somewhat bewildered by the incompleteness of the arguments and the meagre references to actual clinical observation.

The author often fails to clarify his deductions and seems quite unconcerned with any very succinct presentation of facts. But, though the patient reader may often feel that he has been left suspended by a very precarious thread of theory in the somewhat rarefield atmosphere of speculation, he will eventually land on this quite solid ground, namely, that while we have long recognized various vestigial structures in the human organism, obsolete and vestigial functions may continue to be performed and to play a hitherto unrecognized yet vastly important rôle in the reaction of the organism to disease. This indeed may be the foundation of a dynamic pathology.

[1] "Seasonal Variation in Man (A Theory)", by Edmund Hughes; 1931. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 134, with illustrations. Price: 6s. net.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

POST MORTEM EXAMINATIONS.

POST MORTEM examinations may be regarded broadly from two points of view—that of the coroner and that of the medical practitioner. The coroner inquires into deaths which are known to be due or appear likely to be due to violence or accident; and he either gives an order for burial or requires a *post mortem* examination to be made when the deceased person has not been attended by a medical practitioner who can certify to the cause of death. The coroner's function is a protection to the community, but at the same time most valuable information may be gained by his inquiries, especially if the medical practitioners acting on his behalf are not content to fulfil the mere letter of the law, but are imbued with a spirit of scientific inquiry. When a medical practitioner, not acting at the behest of the coroner, undertakes to examine the body of a patient after death, he is searching for information which will help him in his study of disease—he wishes to verify his diagnosis, he seeks for an explanation of obscure symptoms manifested during life, or he inquires into the effects of certain diseases on organs or tissues of the body that he may correlate them with other findings.

Sudden and unexplained deaths are discussed in this issue by Dr. Stratford Sheldon. Dr. Sheldon and Dr. A. A. Palmer, who was associated with him at the meeting of the New South Wales Branch of the British Medical Association, have had a lengthy experience in the investigation of the causes of death in New South Wales, and their words always carry weight. The first point calling for emphasis in Dr. Sheldon's paper is that, although *post mortem* examination may appear unnecessary to determine the cause of death, investigation will sometimes yield surprising information. A second fact is the occurrence of many unexplained deaths. Dr. Sheldon has referred to several deaths which would previously have been regarded as due to *status lymphaticus*. Deaths of this type are constantly recurring. The report of the committee organized by the Medical Research Council of Great Britain and by the Pathological Society of Great Britain to inquire into *status lymphaticus* was discussed in these columns in the issue of July 25, 1931. This committee, composed of distinguished pathologists, concluded that there is no evidence that so-called *status lymphaticus* has any existence as a pathological entity. In our discussion on this subject the statement was made that the onus must be placed on those who believe in a *status lymphaticus* to prove its existence.

Reference to these points in Dr. Sheldon's paper makes it clear that there is urgent need for the wider adoption of *post mortem* examinations. The coroner does not always order a *post mortem* examination to be made. When death of an apparently healthy person is caused by accident, there is no need for *post mortem* examination to determine the cause of death. If, however, examinations were made after all deaths by accident and violence and after all deaths occurring in public hospitals, much would be learned. (In an ideal state autopsies would be conducted after all deaths.) It might, for example, be discovered that the bodily conditions associated with so-called *status lymphaticus* are commoner than is supposed. Apart from this, the correlation of clinical and *post mortem* findings would be of the utmost help in teaching and in diagnosis, prognosis and therapeutics. It should not be difficult to introduce a regulation at teaching hos-

pitals that *post mortem* examinations should be conducted after all deaths occurring at the institution. The first thing to do is to make hospital boards see the necessity for such a step. According to a recent number of *The British Medical Journal*, the British Government has recently declared that the British Post-Graduate Medical School is "an indispensable health service". This attitude should go a long way towards convincing lay boards controlling medical institutions that the acquisition of knowledge is important, that medical practitioners are earnest in their pursuit of it, and that neither sentiment nor need for stringent economy should be allowed to stand in the way.

Current Comment.

UNDULANT FEVER.

A FEW years ago it was believed that undulant fever was confined to the shores of the Mediterranean Sea. Now it is known that the disease occurs in Italy, France, Spain, the countries bordering the Red Sea, India, China, South and West Africa, the West Indies, the Philippine Islands, South America, the United States of America, England and Australia. Within recent years attention has been drawn to the resemblance of the *Micrococcus melitensis* (now called *Brucella melitensis*) to the *Bacillus abortus* of Bang (now called *Brucella abortus*), the causal organism of abortive fever of cattle and pigs. Morphologically and serologically the two organisms are apparently indistinguishable. The question then arose of the pathogenicity of *Brucella abortus* to man. It was found by some observers that man could be inoculated with this organism without any apparent ill effect; on the other hand, there were strong epidemiological grounds for believing that *Brucella abortus* was in some instances the cause of undulant fever, and that the infection could be transmitted in the milk of cows. Bevan, in 1922, expressed the view that infected cow's milk was the cause of an outbreak of undulant fever in Rhodesia. Since then Alice Evans and other observers have found in the blood of persons apparently suffering from undulant fever, an organism morphologically and serologically identical with *Brucella abortus* found in the cow's milk that the patients had been drinking. In May, 1930, Penfold and Butler published in this journal a report of two cases occurring in Victoria, that could be ascribed only to the drinking of infected milk. In February, 1931, Tebbutt and Marsh recorded a case that occurred in the North Coast District of New South Wales; in this instance the patient was a farmer who had been accustomed to handling cattle suffering from abortive fever. *Brucella abortus* may be passed in the urine of dogs, sheep and horses; there is evidence that it may reach the blood of man from this source, by cutaneous inoculation.

There can now be no reasonable doubt that cattle suffering from contagious abortion may be a source of the infection of undulant fever. At the same time it must be accepted that *Brucella abortus* is not always pathogenic to man; apparently it varies considerably in virulence. There is room for doubt whether it is ever equal in virulence to *Brucella melitensis* of goats. It is probable, however, that immunity (natural or acquired) plays an important part in the incidence of undulant fever of bovine origin.

The report of three cases by Edward Dyer Anderson and John F. Pohl is of some interest.[1] Anderson and Pohl's patients were children aged respectively three years, six years and eight years. Many observers have declared that undulant fever is rare among children; but children are not immune. Even suckling infants have been affected. Anderson and Pohl suggest that undulant fever of children may often pass unrecognized. If the disease is common among children there is a ready explanation of the relatively high immunity to the bovine infection among adults.

Each of their patients had been fed with raw milk obtained from a herd of cows among which abortive fever was prevalent. The diagnosis was made by bacteriological investigation in each instance. Two of the patients received milk from the same herd. The most interesting feature in the three cases was the mildness of the symptoms. Practically the only evidence of illness was pyrexia. There were no typical undulations in the temperature chart. In only one instance could enlargement of the spleen be detected by palpation. Diagnosis by purely clinical means was impracticable. The parents of the children were well-to-do and above the average in intelligence, and were all inclined to seek medical advice for very slight ailments. In each instance they had observed no evidence of illness beyond persistent fever. Anderson and Pohl believe that in a less favourable environment the disease must often run its course unsuspected. Less observant parents might easily overlook a fever that causes no obvious symptoms.

A good deal of attention has been paid to undulant fever and abortive fever in the United States of America within recent years. It is safe to say that in conditions such as existed before the disease was recognized in America, undulant fever would not have been suspected. Australia's isolated geographical position guarantees the Australian people a relative freedom from many of the diseases of the old world; but neither this happy isolation nor the vigilance of quarantine authorities can prevent the occasional importation of persons suffering from undulant fever. In 1908 Storie Dixson reported a case in which the patient was a Maltese

[1] *American Journal of Disease of Children*, November, 1931.

immigrant. Doubtless there have been other instances. In many country towns there are conditions favourable to the transmission of the disease by goats. Infected cow's milk must reach thousands of consumers, for abortive fever is common throughout Australia.

Every medical practitioner has occasion to treat persons suffering from fever for which he can find no cause. When such a fever is persistent, the possibility of *Brucella melitensis* or *Brucella abortus* infection might be borne in mind. It seems likely, as Anderson and Pohl suggest in regard to the United States of America, that the disease may often pass unrecognized and unsuspected.

BRONCHIECTASIS.

IT is generally held that bronchiectasis is due to certain mechanical factors acting on a weakened bronchial wall and that in the localized form of bronchiectasis fibrosis plays an important part. M. S. Lloyd has studied the ætiology of bronchiectasis and publishes his views.[1] He holds that the many processes affecting the lung may cause either a temporary or permanent increase or decrease in its volume; and that in bronchiectasis we are concerned only with factors which produce a permanent decrease in volume. Of these factors there are two, atelectasis and chronic inflammation. Since these processes end in fibrosis, "the ultimate cause of pulmonary shrinkage in all cases may be said to be pulmonary cirrhosis". Lloyd's arguments may be summarized in the following way. The first effect of shrinkage of the lung is to increase the intrathoracic negative pressure and exert a traction on all the neighbouring structures in relation with the outside of the closed sphere. It is important to note that Lloyd lays great stress on the recognition of the hemithorax as a unit. If the loss of volume of the lung from shrinkage is slight, the loss may be compensated for by the elasticity of the surrounding lung tissue without the production of any great change in the mechanics of the chest. If, however, the fibrosis has been more extensive, then greater changes must take place. The heart must move toward the affected lung, the diaphragm must rise towards it or the chest wall collapse upon it. If any or all of the adjustments satisfy the pulmonary contraction, further tendency to distortion will be relieved. When a high degree of tension continues to exist, the pathological effects of the imbalance must and will continue.

Lloyd refers to the causes to which bronchial dilatations have been ascribed by others, and names three: (i) pressure from within due to cough or the accumulation of extensive secretions, (ii) chronic inflammations of the lung, (iii) congenital anomalies. He regards the view that pressure from within the bronchi is due to cough as most untenable. Referring to Hedblom's work on intrabronchial pressure, he declares that it is impossible for coughing to produce an intrabronchial pressure greater than the contemporary extrabronchial pressure. He adds that the intrabronchial pressure must always be less than the extrabronchial pressure during coughing, unless the larynx is closed; and under such conditions the intrabronchial pressure may equal the extrabronchial, but can never surpass it.

Lloyd's paper is not very satisfying. He has confined his attention to the purely mechanical aspect of the condition, although he has given his paper the title: "The Etiology of Bronchiectasis." It is true that he refers at the beginning of his discussion to atelectasis and chronic inflammation as resulting in fibrosis, but there he leaves them. He makes no mention of the effect of infecting organisms on either the cells lining the bronchi or on the muscular tissue outside them. No discussion on the causation of bronchiectasis can be complete which does not deal with the early changes. In many instances there is no doubt that the constant reinfection of the bronchi from the upper respiratory passages is the underlying cause. Once fibrosis has started, a sort of vicious circle is established. Much that Lloyd has written about the mechanism of bronchiectasis is probably correct, but he has not proved that pathologists such as Muir are mistaken in the view that the chief mechanical factor in dilating the bronchi is forced inspiration, especially that which follows the act of coughing—that excess of air being suddenly drawn into the bronchi, brings about their dilatation.

[1] *The New England Journal of Medicine*, December 10, 1931.

THE LEAGUE OF NATIONS HEALTH ORGANIZATION.

IN 1924 the League of Nations Health Organization established at Singapore an epidemiological bureau intended to fulfil two major purposes. It was to serve as a centre for the dissemination of information concerning the occurrence and progress of the principal epidemiological diseases and it was to coordinate the research being carried out by the different national organizations. This bureau has been working to the satisfaction of all concerned since its inception, especially in regard to the first of its two functions. A weekly message is broadcasted from the Saigon radio station, which gives to all stations from Vladivostok to Wellington and Hong-Kong to Port Said an exact account of the condition of plague, cholera and smallpox in each of the ports in that area up to noon on the preceding Saturday. At the outset the League appointed eight persons representing the different countries as members of an advisory council. A special meeting of this advisory council is being held by invitation of the Japanese Government in April next for the purpose of reviewing the progress made and of examining the possibilities of extending the work in other directions. Dr. J. H. L. Cumpston, Director-General of Health of the Commonwealth, will represent Australia at the meeting.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

The Problem of Sepsis.

P. SAXL (*Wiener Medizinische Wochenschrift*, February 28, 1931) reviews in detail the problem of septic infection, with special reference to the streptococcus. He is of the opinion that the streptococcus, especially the *viridans* variety, can change its characteristics and that owing to acquired immunity of the body the virulence may be decreased. Apart from the work of Dick on the scarlet fever toxin, no similar results have been obtained for other streptococcal infections, possibly because the most virulent develop little toxin. The question of localization of infection has been complicated by the work of Rosenow. Previously sepsis was believed to be a localized or general infection, but Rosenow has introduced a third group of elective localization with focal infection, principally from the tonsils or teeth as the main source of the organisms. As regards immunity, mixed infection plays an important rôle. In the treatment of septic conditions the author considers that vaccine therapy is useless. The antivirus treatment of Besredka with filtered cultures of dead organisms has shown some success, as has also serum therapy. He is more in favour of non-specific treatment with the aniline dyes and collargol.

Occupation and Serological Reactions for Brucella Abortus.

AXEL THOMSEN (*Journal of Infectious Diseases*, May, 1931) discusses the correlation of occupation with serological reactions for *Brucella abortus*. He noted a striking discrepancy between cases of undulant fever reported among veterinarians in Denmark and the marked degree to which workers in this field are exposed to infection when delivering the placenta in aborting cows and assisting at births. He sought to prove that an immunity must account for this discrepancy by searching for demonstrable antibodies by agglutination and complement fixation tests. In all, two hundred and seventy-two persons in various occupations associated with cattle were studied, and sixty-one healthy persons in other occupations served as controls. The results are summarized as follows. Of sixty-one veterinary surgeons in rural practice for more than one year, 94% gave positive reactions. The results of complement fixation tests were more often positive than those of agglutination tests. An entire class of young veterinarians tested before their final examinations gave no reaction. The members of another entire class, eighteen in all, were examined five months after leaving school; fifteen reacted, with titres equal to those seen in cases of undulant fever. Only one had clinical undulant fever. Members of a third class, examined about one year after leaving school, gave reactions correlating with those of the preceding group. Those who did not give reactions were not handling cattle. Of sixteen bacteriologists working with *Brucella abortus*, ten gave positive reactions (62%). Of twenty-one chief cattle attendants at farms where contagious abortion existed, six gave positive results (24%). Of twenty-three owners of such farms, nine gave positive reactions. Among twenty milkmaids at similar farms, only one gave a reaction. No reactions were noted among ten milk tasters. Of five inspectors of milk and stables, one gave a positive reaction. This man had formerly engaged in rural practice. Five of twenty-five butchers gave positive reactions. Of twelve inspecting veterinarians examined at slaughter houses, four yielded positive sera; all four had formerly practised among cattle. It would appear, therefore, that interpretation of positive results in diagnostic serological tests among those in occupations connected with cattle or with *Brucella abortus* should be made with care, since the sera of healthy persons thus employed may give positive results.

The Skin as an Immunological Organ.

LOUIS TUFT (*Journal of Immunology*, August, 1931), during the course of some experiments in serum sickness, observed that the skin seemed to possess an unusual capacity for sensitization to horse serum, this sensitization being accompanied by the production of anaphylactic antibodies. These experiments also suggested the possibility that the skin might act as an important immunological organ. A review of the literature showed that this conception of the skin has been gaining increasing recognition and is supported by many clinical facts. For instance, with the acute exanthematous diseases a marked skin eruption is considered of good prognostic import, and patients with skin tuberculosis rarely develop pulmonary involvement. A comparative study of the antibodies produced after subcutaneous, intradermal and intramuscular administration of mixed typhoid vaccine was carried out. It was found that with the intradermal method, in spite of the fact that the dose was one-seventh the size used in the other methods, the antibody response was equal to that produced by the subcutaneous, and slightly better than that produced by the intramuscular method. Eight individuals were injected by the intravenous method, and the antibody response was not quite so good nor so persistent as by the other methods. There seemed to be a tendency for rapid but temporary stimulation of antibody after the intravenous injection. It would appear from this work that in man better and more persistent response is afforded by injection of typhoid vaccine attended by local reaction, particularly when the skin is involved in the reaction, than when the vaccine is given intravenously. Thus antibody formation in relation to bacterial infection is not a property specifically of one organ, but rather of a tissue present in many organs, and this tissue is represented by the reticulo-endothelial system. The author holds that this conception of the skin as an important immunological organ explains in part the satisfactory results of non-specific mechanical therapeutic measures employing the skin surface for stimulation, and suggests the possibility of utilizing the skin therapeutically for measures of specific active immunization in infectious and allergic diseases. It also substantiates the rationale of the various methods of cutanotherapy advised by the French and Germans, especially Besredka.

The Specificity of Avian Tuberculin Reactions.

VERA B. DOLGOPOL (*Journal of Infectious Diseases*, September, 1931) inoculated sixteen chickens with material from patients suffering from advanced pulmonary tuberculosis and showing positive reactions with avian tuberculin, and twelve chickens with material from patients not reacting to avian tuberculin, and eight chickens with material from untested persons. In all cases the material contained large numbers of acid-fast bacilli. In none of the chickens that received the material, either intravenously or intraperitoneally, did tuberculosis develop. From these results it is concluded that a positive reaction to avian tuberculin observed in many cases of pulmonary tuberculosis does not indicate an active mixed infection with human and avian tubercle bacilli or latent infection with the avian tubercle bacillus; and that the large percentage of positive reactions with avian tuberculin in tuberculous patients must be considered only as a large percentage of group reactions in patients suffering from tuberculosis caused by the mammalian types of tubercle bacilli. A positive reaction with avian tuberculin in the presence of positive cutaneous reactions with mammalian tuberculin does not indicate an infection with the avian tubercle bacillus, but such an infection may be assumed to exist if the reaction with avian tuberculin is positive while the reactions with other tuberculins are absent or considerably weaker.

The Gonococcus.

L. T. CLARK, N. S. FERRY AND A. H. STEELE (*Journal of Immunology*, September, 1931) have prepared and tested a toxic filtrate from liquid culture of the gonococcus following a request for a bouillon filtrate of the gonococcus in the nature of an antivirus. A liquid culture was incubated for seven days only to eliminate as far as possible autolytic products which are known to be toxic and to occur easily in gonococcal cultures. Tests of this filtrate showed that it

was not only toxic, but antigenic as well, and still permitted a luxuriant growth of the organisms if reseeded; this differentiated it from an antivirus. The media used and precautions taken to prevent autolysis are described in detail. Doses of the filtrate, diluted 500 to 1,500 times, gave typical local reactions when injected intradermally into 300 individuals. This skin reaction varied in intensity, reached its maximum in about twenty-four hours, and then rapidly subsided. No reactions were observed in certain individuals, but whether this indicated an immunity to gonococcal infection was not disclosed. No relationship was found between these skin reactions and recent, remote or chronic infections. The filtrate was found not to be uniformly toxic to laboratory animals. Injection of animals with the filtrate stimulated the formation of an antitoxin which neutralized the toxin *in vitro* as well as *in vivo*.

HYGIENE.

Dangers in Refining Radio-Active Substances.

H. SCHLUNDT, W. MCGAVOCK AND M. BROWN (*Journal of Industrial Hygiene*, April, 1931) have investigated the hazards involved in the refining of commercial mesothorium. The investigation was conducted along three lines: the quantitative measurement of the intensity of radiations remaining in the laboratory after precautions had been taken to reduce them to a minimum was carried out; the workers were examined for accumulated active material and a record was kept of the physiological and cytological conditions. A description of the technical methods used in the first portion of the inquiry is given. The workers, three in number, who worked in the laboratory from fifteen to thirty hours a week, were examined electroscopically following a day's absence, with a bath and hair shampoo. It was found that the workers were inhaling or absorbing about 2% of a dose of radium, just within the limits of tolerance. The laboratory air contained radon and thoron in amounts considerably above those of the ordinary atmosphere. Thoron was much more abundant than radon. The blood of the workers showed no abnormality, and no debilitating effects have been observed.

Coal Miner's Lung.

THE King Edward VII Welsh National Memorial Association (*Journal of Industrial Hygiene*, January, 1931) conducted a study of miners employed in getting three different types of coal. As a comparison, a series of twelve radiographs of the chests of healthy agricultural workers was obtained. The investigation was conducted by three teams of medical men, each of which worked on its own lines. Miners of fifteen years' or more experience who had not worked in hard headings were examined. The first team worked in an area in which the type of coal is submetabituminous and orthocarbonaceous, and the country rock shales and beds of sandstone. Abnormal X ray appearances were found in 26 in a series of 48 coal hewers. The second team worked in an area similar to number 1 team. Forty-one men were examined, of whom 21 showed radiographic abnormalities. The third team examined 42 workers in anthracite coal, which outcrops in the district. By a system of graduation of mottling and estimation of the result as a percentage, over 70% of mottling was observed in these men. The conclusions are drawn that prolonged exposure to the working conditions of coal mining is associated with production of changes in radiographic appearances of the lungs, and that these changes are manifest as fine or coarse mottling comparable to that seen in silicosis. The changes increase in amount with duration of employment, and represent definite impairment of lung structure.

Linseed Dermatitis.

M. H. BARNES (*Journal of Industrial Hygiene*, February, 1931), after observing that the labour turnover in the flax seed industry is very large, has made a study of the action of the seed and its products on the skin of workers. It appears that certain individuals react without previous sensitization to some irritant that linseed cake contains, and that this irritant is not present in pure linseed oil or in impurities removed from linseed by passing it through sieves.

Institutional Treatment for the Tuberculous.

JAMES WATT (*Journal of State Medicine*, June, 1931) traces the development of sanatoria and the institutional treatment of tuberculosis from its inception in England thirty years ago, pointing out its educative value and the limitations of home treatment. The sanatorium must be considered as a repair depôt for patients in any stage of the disease. The X ray installation is of the greatest importance. The routine supervision of contacts will reveal a definite percentage of early cases, much higher than that of the general population. At all institutions observation beds should be provided. If these facilities for early diagnosis were universally available, the results of treatment would be much improved. Dispensaries should be attached as out-patient branches to general hospitals. In-patient treatment should be continued no longer than is strictly necessary, and personal subsidies to needy patients should be paid on condition that they undergo suitable institutional treatment. Regarding internal sanatorium administration, the first point to be stressed is the education of the patient to enable him to lead the correct life outside and avoid infecting others. Patients should stay a sufficient time under treatment to enable their discharge as sputum-free, or at least with sputum free of tubercle bacilli for three months. Great tact on the part of the physician is necessary, and, when possible, active treatment, not of the placebo type, is helpful in this direction, giving encouragement to the patient. The appointment of a sanatorium medical officer as tuberculosis officer in the district is advantageous if it can be accomplished without understaffing, as the viewpoint of the officer is thereby widened. In hospitals for advanced and chronic cases there should not be too many deaths; all sanatorium treatment provisions should be used; occupational therapy should be provided; large wards are preferable to single room accommodation; and there should be a dilution with ambulant and even early cases and free transfer to sanatoria of improving patients. The provision of institutional treatment for the dying is a great problem. The increased infectivity of such patients renders institutional treatment desirable. The tuberculosis hospital should take its share of these cases, but the bulk naturally falls to the general hospital. Patients with doubtful or mild disease who object to sanatorium treatment, might now be treated under expert supervision in a convalescent home. The institutional treatment of children is complicated by the large numbers of doubtful cases. Patients suffering from non-pulmonary disease should be treated in special institutions in the country or at the seaside. Village settlement schemes represent the most controversial subject, on account of the large initial expenditure, the need for subsidization of industries, and the limited numbers of people likely to benefit. Consequently, their multiplication remains a matter of doubt.

Sex Differences in the Physical Impairments of Adults.

ROLLO H. BRITTEN (*American Journal of Hygiene*, May, 1931) compares the results of medical examinations of male and female adults by the Life Extension Institute of the United States of America. The group probably includes too small a proportion of the lower social and economic grades to render the results absolutely representative of the nation, and the fact that persons volunteer for examination because they consider they have something wrong will further tend to the production of minimal rates. The age groups for the two sexes are almost identical in proportion. Examinations performed at central offices are tabulated separately from those performed at outlying branches, and the results point to the former being more thorough. Comparison of female with male conditions shows a large preponderance among females of thyreoid troubles. The average rate for all diseases is higher among females than males. The rates for males for casts in the urine, deflected septum and arterial thickening are higher than for females. The age curves for the two sexes are generally identical.

Special Articles on Aids to Diagnosis.

(Contributed by Request.)

XIV.

THE DETERMINATION OF RENAL EFFICIENCY BY ESTIMATION OF BLOOD UREA AND THE UREA CONCENTRATION TEST.

WHILST numerous tests have been suggested and are used for the determination of renal efficiency, no combination of methods has perhaps become more popular than that of the estimation of blood urea coupled with the urea concentration test. Both tests have certain limitations, but when they are used as adjuncts, most valuable information concerning renal function may be obtained.

Blood Urea.

Urea is constantly formed in the body as the result of the katabolism of proteins and is just as constantly excreted by the kidneys, hence the percentage of urea in the blood at any moment represents a balance between its production and its elimination. Urea filtered through the glomeruli is not absorbed in the tubules, but water is rapidly reabsorbed, so that the concentration of urea becomes more marked the further the glomerular filtrate passes along the tubules. A 5% solution represents the approximate limit of the kidney's power to concentrate urea in the urine, so that an adequate supply of water is necessary in order that the urea formed in the body may be eliminated. If sufficient water is not available, then urea retention will inevitably occur, even though the kidneys may be quite normal.

In health, the amount of urea found in the blood when the patient is on an ordinary diet, varies from 20 to 40 milligrammes per 100 cubic centimetres. In clinical work figures above 40 milligrammes are usually regarded as abnormally high. Small variations in the diet do not appreciably influence the blood urea, but marked limitation of the protein intake may be responsible for the figure falling as low as 10 milligrammes per 100 cubic centimetres. On the contrary gross overfeeding with protein may raise the urea to 50 milligrammes.

During the later stages of pregnancy the nutritional demands of the fœtus may be responsible for diminution of the amino acid content of the maternal blood, and the blood urea which ultimately owes its origin to these protein derivatives, may be markedly decreased. In the normal individual about one-fourth of the total renal tissue is capable of performing the necessary excretion. Hence, if unilateral nephrectomy be performed, the blood urea will not show any subsequent rise provided the remaining kidney is approximately normal. In patients whose kidneys are severely damaged, the quantity of urea excreted per day may be normal, but in such circumstances the concentration of urea in the blood may be greatly increased, amounting perhaps to 200 milligrammes or more per 100 cubic centimetres. Owing to the ready solubility of urea and its marked diffusibility, it is found in equal quantities in plasma and in corpuscles, hence whole blood, plasma or serum may be used for analysis.

Methods of Estimating Blood Urea.

1. *Hypobromite Method.*—The proteins in a measured quantity of blood are precipitated by trichloracetic acid and an aliquot part of the protein-free filtrate is treated with sodium hypobromite. The nitrogen liberated is measured and the percentage of urea calculated. The method is not so accurate as the urease method about to be described, and is seldom used in clinical work.

2. *Urease Method.*—The estimation of the amount of urea in blood is based on the conversion of urea into ammonium carbonate by the ferment urease. Ammonia is liberated from the ammonium carbonate by the addition of potassium carbonate and is then estimated either colorimetrically after Nesslerization, or by titration after aeration into standard acid, as in Maclean's modification of the method introduced by Marshall, Folin and van Slyke. Maclean's method will now be described in detail.

The reagents necessary are: (i) Potassium oxalate (20%), (ii) sulphuric acid (5%), (iii) acid potassium phosphate (0·6%), (iv) capryl alcohol, (v) soya bean meal, (vi) sulphuric acid, a one-hundredth normal solution, (vii) sodium hydroxide, a one-hundredth normal, (viii) methyl red (saturated solution in 50% alcohol), (ix) solid anhydrous potassium carbonate, (x) saturated solution of potassium carbonate.

About eight cubic centimetres of blood are withdrawn from a vein at the bend of the elbow, coagulation being prevented by the use of a small amount of finely powdered potassium oxalate, or one drop of a 20% potassium oxalate solution.

A wash bottle, A, and two large test tubes, B and C, about 200 millimetres deep and 25 millimetres wide, with well-fitting rubber corks, are required (see Figure I). Through each rubber cork two glass tubes pass, one of which is long and dips into the liquid contained in the respective test tubes; that in C has a perforated bulb on the lower end. The short tubes pass just through each cork. The test tubes and wash bottle are connected together by means of tubing having suitable clips, E and F, attached.

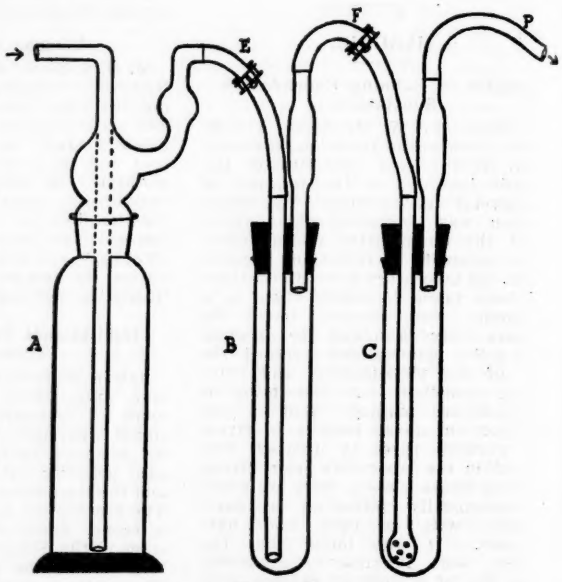

FIGURE I.
Showing arrangement of apparatus for estimation of blood urea.

Into the wash bottle A are introduced about 20 cubic centimetres of a 5% sulphuric acid solution. Into B five cubic centimetres of 0·6% acid potassium phosphate solution are measured, and by means of a special pipette three cubic centimetres of blood are introduced, and the pipette thoroughly washed out two or three times with the phosphate mixture so as to remove all traces of blood. From six to eight drops of capryl alcohol are added and, finally, 0·3 gramme of ground bean. Freshly ground beans should be used, as it is found that old preparations may yield considerable quantities of ammonia, which vitiate the result. The variety of soya bean known as Ito San appears reliable, but in hot climates the jack bean is preferable, as it has less tendency to liberate ammonia. The rubber stopper is quickly replaced and the tube closed by means of the clips on the rubber tubing. The tube is placed in a water bath at a temperature of 40° to 45° C. and left for fifteen minutes with occasional shaking. At the end of this time the tube B is removed from the water bath and connected on the one side with the wash bottle

and on the other with C, which contains 25 cubic centimetres of one-hundredth normal sulphuric acid solution, one drop of capryl alcohol and two drops of methyl red solution. The short exit tube of C is connected by pressure tubing to a water suction pump.

The clips are now opened and air is drawn slowly through the whole system for two minutes. The air entering A has all traces of ammonia removed by sulphuric acid. It then passes through the blood in B and finally through one-hundredth normal acid in C, which takes up any ammonia carried from the blood. After two minutes suction is stopped, the blood tube B opened and four cubic centimetres of saturated potassium carbonate solution followed by three grammes of solid anhydrous potassium carbonate, are quickly added. The stopper is immediately replaced and a current of air drawn through, at first slowly and then rapidly. The ammonia should be completely removed in about thirty minutes.

The tube C, containing the standard acid, is now disconnected and the acid transferred quantitatively to a small Erlenmeyer flask. The perforated bulb is thoroughly washed two or three times in distilled water.

The acid is then titrated with one-hundredth normal sodium hydroxide solution until the indicator gives a faint yellow colour.

The difference between the 25 cubic centimetres of acid originally taken and the number of cubic centimetres of alkali used gives the number of cubic centimetres neutralized by ammonia. From this 0·4 cubic centimetre must be subtracted to allow for traces of ammonia present in the soya bean meal. If the above quantities are used, each cubic centimetre of acid neutralized equals 10 milligrammes of urea per 100 cubic centimetres of blood.

The Clinical Value of Blood Urea Estimation.

In Medicine.—The chief conditions in which urea may accumulate in the blood are acute and subacute glomerulonephritis, renal tuberculosis and polycystic disease of the kidneys.

In the acute and subacute forms of nephritis the increase of blood urea may be due in part to diminished quantity of water excreted and is promptly relieved if diuresis occurs, hence in these conditions the prognosis based on blood analysis is a little uncertain, except in patients who show very high urea—200 to 400 milligrammes of urea per 100 cubic centimetres, when the outcome is usually fatal.

A moderately raised blood urea—100 milligrammes per 100 cubic centimetres—is not usually of such serious moment in acute nephritis as a similar finding in a case of chronic interstitial (azotæmic) nephritis. In the former the tendency to recover is frequently very considerable, whilst in the latter the lesion is usually progressive, hence the prognosis in any patient should be judged in the light of his history, physical examination and other renal tests and not on the blood analysis alone. Nevertheless, estimations of the blood urea every few days during the course of acute nephritis are of considerable value in determining the progress of the disease.

In interstitial nephritis and in polycystic kidney disease, polyuria occurs, so that any retention of urea is probably due to renal inefficiency and not to inadequate excretion of water, which might, as previously explained, interfere with the urea output. It is the general consensus of opinion that in these chronic renal conditions a blood urea of 200 milligrammes or more indicates that death will occur within six months. With a blood urea of 100 milligrammes the duration of life will probably be less than two years, whilst with minor rises in the blood urea the prognosis is indeterminate. It is well to remember that a nephritic patient who has been on a very low protein diet for some months may have a comparatively normal blood urea, though he may be on the verge of uræmia. In such circumstances a urea concentration test would reveal poor concentration powers and the true state of renal function. In any case it is unwise to base one's opinion on a single blood analysis. At least two or preferably more should be made at an interval of about a month.

Many conditions other than renal disease may be associated with a raised blood urea and the symptoms simulate uræmia. It is not uncommon for this to occur in intestinal obstruction, diabetic coma, gastro-enteritis or general peritonitis. These conditions, however, are all characterized by desiccation of the body due to deficient intake of fluid and excessive loss of such. In these circumstances the rise of blood urea is simply due to the desiccation of the body. The urea concentration test will enable the physician to absolve the kidneys, for if the condition is merely one of retention of the urea due to desiccation, then the kidneys will be able to concentrate urea in the urine even to 3% or more. Such concentrating powers do not occur in uræmia.

The dyspnœa and œdema which may accompany cardiac failure due to arterial disease and high blood pressure are sometimes erroneously diagnosed as of renal origin. If the blood urea percentage is found to be approximately normal and the urea concentration test shows a figure between 2% and 4%, the patient's symptoms are probably cardio-vascular in origin, and renal disease can be eliminated as the chief cause.

In Surgery.—In prostatic disease the renal impairment is in part caused by the back pressure of retained urine, and suitable drainage may greatly improve the condition. If the blood urea exceeds 50 milligrammes per 100 cubic centimetres, a two-stage operation is usually indicated. Following suprapubic cystotomy and drainage of the bladder, considerable amelioration of the renal function may occur, as indicated by a diminution of the blood urea and an increase in the well-being of the patient.

In renal tuberculosis or calculus pyelitis where nephrectomy is contemplated, it is wise to estimate the blood urea which, if raised, indicates impairment of the function of the second kidney, since one healthy kidney is more than adequate to keep the blood urea at a normal level. The relative efficiency of the kidneys may also be determined by the urea concentration test, separate samples of urine from each kidney being obtained by catheterization of the ureters.

In Obstetrics.—In nephritic toxæmia of pregnancy some advise that the pregnancy should be terminated if the blood urea rises above 40 milligrammes per 100 cubic centimetres, but the wisdom of such a procedure must be largely determined by the result of the general examination of the patient and the confirmation of renal impairment by other tests. To allow such a patient to proceed to term is stated to aggravate the preexisting renal lesion, so that the renal efficiency is much worse after delivery than before. In preeclamptic toxæmia the blood urea may or may not be raised, but in eclampsia a rise is of common occurrence.

The Urea Concentration Test.

Since normal renal excretion may be maintained when approximately 25% only of the kidney substance is functioning, it is obvious that very considerable pathological changes may occur in the kidneys before inadequacy is revealed by such a quantitative method as the estimation of blood urea. When, however, the margin of physiological reserve is reached, these organs fail to concentrate satisfactorily in the urine a substance presented to them in dilute solution in the blood. This failure to concentrate is an earlier sign of renal disease than is the retention in the blood of the waste products of nitrogenous katabolism such as urea, uric acid and creatinine. In Maclean's test a measured quantity of urea in aqueous solution is administered by mouth and the concentration of urea in the urine at the end of each of the following three hours is used to indicate the functional efficiency of the kidneys. The test is so simple in technique that it should be performed by practitioners on all patients exhibiting albuminuria or other evidence of renal pathological change.

Procedure.

Administration of Urea.—It is usually most convenient to perform the test in the morning, the patient having fasted for twelve hours and had nothing to drink. At 7 a.m. he passes his urine and immediately takes by mouth 15 grammes of urea dissolved in 100 cubic centimetres of water and flavoured with one cubic centimetre of tincture

of orange. At 8 a.m. the bladder is emptied and the entire specimen kept. Similarly, at 9 a.m. and 10 a.m. the patient passes his urine, completely emptying the bladder on each occasion. These three specimens are labelled and the volume of each is measured and the percentage of urea determined by the hypobromite method.

Estimation of Urea in Urine.—The apparatus (see Figure II) should first be tested for leaks by closing the stop-cock C and developing pressure by raising the levelling tube E. About 10 cubic centimetres of sodium hypobromite are introduced into the bottle A, and two cubic centimetres of urine are measured very accurately

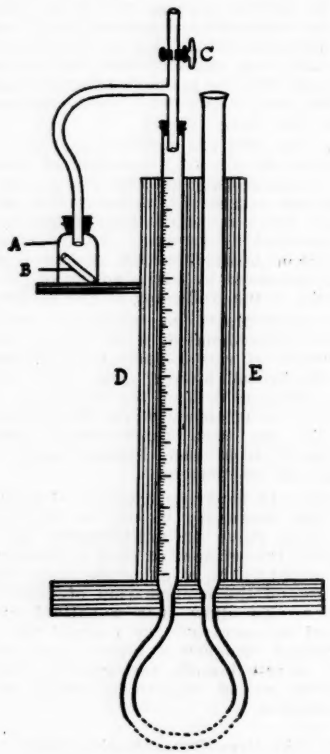

FIGURE II.

Apparatus for estimating the percentage of urea in urine.

into the small tube B. The latter is now carefully lowered into the bottle A, the cork is tightly inserted and the bottle placed in a water bath. With the stopcock open, the level of the water is brought to zero and the stopcock then closed. The urine is now mixed with the hypobromite by tilting the bottle several times. The evolution of nitrogen is accompanied by the production of considerable heat, and the bottle is replaced in the water bath and allowed to cool, when the menisci of the measuring burette and tube E are brought to the same level and the volume of nitrogen eliminated is read.

Calculation.—One gramme of urea evolves 354 cubic centimetres of nitrogen. If X be the volume in cubic centimetres of nitrogen liberated, then

$$\frac{X}{354} \times \frac{100}{2} = \text{percentage of urea in the urine.}$$

Tables have been prepared showing the percentage of urea equivalent to the number of cubic centimetres of nitrogen evolved.

Clinical Value of the Test.

In health the urea concentration following Maclean's test should be at least 2% and the normal kidneys frequently excrete a urine containing 3% to 4% of urea. Less than 2% indicates renal impairment, and the lower the figure the graver the outlook.

It must be emphasized that this test is not used to determine the total output of urea in a given time, but rather the ability of the kidney to concentrate urea in the urine. Diuresis usually follows the administration of the urea, and if this results in an output of more than 120 cubic centimetres in the first hour, or more than 100 cubic centimetres in the second or third hour, then a low percentage of urea might be due simply to the large volume of urine excreted, and not to failure of concentrating power of the kidneys. Under these conditions it is wise to repeat the test in the hope that the volume of urine excreted on the next occasion may approximate more closely to that which has been arbitrarily fixed as normal. Since diuresis frequently passes off during the first hour, most authorities prefer to examine only the second and third hour specimens and to base their opinion on these estimations.

Frequently in hospital reports the percentage of urea is stated without the figures being supplied, indicating the corresponding volumes of urine. Fallacious deductions may readily be made in such cases.

Reference has already been made to the value of the urea concentration test in distinguishing high blood pressure due to vascular disease from that due to renal disease. The use of the test in distinguishing uræmia from conditions of non-renal origin but with raised blood urea must again be stressed. The test, however, frequently fails to indicate renal impairment which may exist in hydræmic nephritis with chloride retention and a normal blood urea.

In surgery the urea concentration test has a special application in determining the relative efficiency of the two kidneys, the ureters being catheterized to obtain the separate specimens of urine.

In a recent communication on the chemistry of blood and urine in toxæmias of pregnancy Krieger and Green state that in these conditions the urea concentration test is more discriminating and more useful than the estimation of blood urea. They, however, relied more on clinical judgement than on biochemical tests, but the urea concentration often provided useful confirmation of their judgement. In a doubtful case a low urea concentration figure (for example, 1·5%) on two occasions implied unsatisfactory response to treatment and indicated the necessity for induction of labour.

From the foregoing review it will be realized that the estimation of blood urea and the performance of the urea concentration test have many valuable applications for the practitioner, and in conclusion one would suggest that if these two methods of bio-chemical investigation were more widely used by physicians, surgeons and obstetricians, greater accuracy in diagnosis and increased success in treatment of renal conditions would result.

IVAN MAXWELL, M.D., M.Sc.,
Lecturer in Physiology and Clinical Biochemistry, University of Melbourne; Physician to Out-Patients, Melbourne Hospital.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on November 26, 1931, DR. ST. J. POOLE, the Vice-President, in the chair.

Child Welfare Problems.

DR. HELEN MAYO read a paper entitled: "Problems in the Working of a Child Welfare Scheme in the Community" (see page 249).

DR. PERCIVAL CHERRY said that he appreciated Dr. Mayo's paper, and assured her that in his district, at least, the Children's Welfare Branch was doing a great amount of good. In Port Adelaide, where about half the population was on Government relief, there were frequent demands for fresh milk, an item not on the usual ration scale. The School for Mothers had frequently refused this request, pointing out that three good meals a day for the nursing mother, with plenty of water as fluid intake, would do as much or more good than indulgence in milk and the usual "slop" foods. It had been shown that the calorific value of the ordinary adult "ration" was very generous for ordinary requirements and quite sufficient for lactation, which, after all, was only an ordinary physiological and not a pathological process. The babies in the Port Adelaide district, as a result, were just as healthy as their brothers and sisters used to be, and as robust as infants elsewhere.

Occasionally a "welfare" nurse might overstep her mark. A few days previously an infant was brought by its mother to him to have its tongue cut. The child was splendidly nourished, so apparently could take the breast or bottle quite naturally, in spite of a possibly (only) slightly shortened frænum. The tongue cutting request was not granted.

As an offset to this, one must not always take the parent's word against the nurse, as very frequently the nurse was misreported or her actual statements misinterpreted.

DR. E. A. H. RUSSELL thanked Dr. Helen Mayo for her excellent paper. He thought, however, that closer supervision should be kept over the nurses attending the baby health centres. He had found that in some instances the whole scheme of feeding an infant that he had advised had been entirely altered by the nurse at the baby health centre and some other scheme adopted. This was a very improper and unethical procedure that created an atmosphere detrimental to all.

Another difficulty often experienced was that mothers sometimes could not or would not go out regularly to have their babies weighed. Adverse weather conditions, illness in the house, and sometimes visitors, were quite sufficient to keep the mother and baby at home. The result was a break of two, three, or even four weeks in registering the weight of the infant. To overcome this he had for many years encouraged the mother to have in her own home suitable clock-faced scales. In practice, even the cheaper scales were sufficiently reliable for the average mother to form a fairly accurate idea as to her infant's progress.

The mother was instructed to weigh the child every week, on the same day of the week, at the same time of the day (just prior to the second feed), in the nude, and to keep a record of the weight on a chart. If the weight was not satisfactory, the mother brought the chart and the infant to the surgery, where a closer supervision could be kept and the necessary treatment instituted.

DR. R. G. BURNARD said that his relationships with the baby welfare centre had always been most amicable. At the same time, he thought it essential that the general practitioner should make himself proficient in all knowledge pertaining to infant welfare, so that the nurses at the centre should be able to cooperate with him in confidence. He encouraged the mothers to bring their babies to his surgery for observation and advice. He thought that a correct baby scale, capable of weighing fractions of ounces, was an essential part of the equipment of the general practitioner.

Dr. Mayo, in reply, said that though it was almost inevitable that mothers would return to maternity nurses or homes for advice, the practice was subject to grave abuses and should not be encouraged; organized and supervised work like that of the welfare centres was more easily controlled.

The problem of the ancillary services was by no means confined to nurses, as they all knew. This was exemplified in a pharmaceutical journal recently, which quoted the case of a dentist who had, against the medical practitioner's advice, insisted on colonic wash-outs for a patient, and held that the result justified his action.

Dr. Hone's suggestion for a book for all babies, in which medical attendant and nurse wrote their directions, was a good idea, if practicable, being an extension of the present card system. His suggestion of a circular to all members of the Branch should certainly be adopted.

With regard to Dr. Gilbert's remark that it was difficult to retain the milk supply on four-hourly feeds, it was usually necessary to give both breasts at each feed. When this was done, most babies did well and the supply kept up.

With regard to the assertion that the nurses took the babies off the breast, Dr. Mayo was quite sure that this was not true; it must be due to a misunderstanding. One of the complaints that the nurses most frequently brought against the medical practitioners, was that they weaned the babies that were sent to them—no doubt also a misunderstanding.

What constantly happened was that the mother did what she wanted to do and then accused doctor or nurse of having ordered it. No satisfactory test weighing could be done on spring scales.

Dr. Mayo asked medical practitioners when they received a note from the nurse asking them to see a baby, to accept it as a friendly act and to uphold the nurse. If they discredited her action, and it was very easy to do this by the merest gesture, it would be much more difficult for her to persuade the mother to come to the medical practitioner when next the occasion arose. The task of persuading a mother to take her baby to the medical practitioner was often far from easy, and if they did not cooperate it might be impossible.

Dr. Mayo was sure that this discussion was the beginning of a much better understanding and she thanked members for coming and taking part.

Electrocardiography.

DR. E. F. GARTRELL exhibited a portable electrocardiograph instrument which he had had made in Adelaide. He said the Cambridge instrument, which embodied a string galvanometer, was both too cumbersome and too fragile to be moved from house to house. As many occasions had arisen when an electrocardiogram was most desirable and when the patient was too ill to be moved, it was obvious that this shortcoming was a serious one.

The main principle of the new instrument was the amplification of the heart current by means of valves wired in a special manner. The amplified current was passed through a steel bar in a magnetic field, this bar supporting a small mirror. When the current passed through and so deflected the bar, it also moved the mirror which in turn deflected a beam of light whose movements were recorded photographically.

Dr. Gartrell placed a patient in circuit and demonstrated the simplicity with which the instrument could be manipulated.

Intratracheal Ether Apparatus.

DR. E. COUPER BLACK showed a new apparatus to vaporize ether for intratracheal or intrapharyngeal anæsthesia. The principle was that an outer compartment containing hot water surrounded an inner one through which flowed a current of air from an electrically driven pump. At a rate controlled by a needle valve ether dropped into the warm air stream and was vaporized immediately, thus giving very fine gradations in the percentage of ether suitable for a baby, child or adult. The rate of the drops could be seen through a little window. The ether was fed up to the dropper from an ordinary bottle, fitted with a rubber cork and two glass tubes, by means of positive pressure on its surface, the flow being started and maintained by occasional use of a hard bellows connected to the bottle. The apparatus was modelled on the ideas of Shipway and Magill. It was made by W. Rogers, of Adelaide, was small and compact, and the workmanship compared more than favourably with such apparatus made abroad.

Carcinoma of the Œsophagus.

DR. GLYNN showed the radiograms of a case of carcinoma of the cardiac end of the œsophagus treated by insertion of a Souttar's tube. He considered this the best palliative

treatment of the condition. He had had one fatality, but in the other cases no ill effects had occurred. The patients were freed of the pseudo-vomiting which persisted even after gastrostomy and, providing they chewed their food well, could eat most things.

Vesicular Dermatitis.

DR. IAN HAMILTON reported a case of vesicular dermatitis. This report will be published in another issue.

Fibromyoma of the Uterus.

Dr. Hamilton also reported a case of fibromyoma of the uterus which had given rise to intraperitoneal hæmorrhage. This report was published in the issue of February 13, 1932.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, 137, Macquarie Street, Sydney, on November 26, 1931, DR. GEORGE BELL, the President, in the chair.

Sudden and Uncertified Deaths.

DR. STRATFORD SHELDON read a paper entitled: "Sudden and Uncertified Deaths" (see page 252).

DR. A. A. PALMER gave a demonstration of firearms and described their mechanism. He showed that discharge of a firearm, such as a revolver or a police automatic pistol, caused indentations in the cartridge case and on the bullet peculiar to each weapon. He showed photographs taken by officers of the Police Department of New South Wales to illustrate his remarks. He maintained that, considering the small amount of available material, work of a high order was being carried out in Sydney.

Referring to Dr. Sheldon's paper, Dr. Palmer said that sudden death from interference with the uterus was not always due to air embolism, but could be caused by shock.

Dr. Palmer spoke of sudden death resulting in several instances from hydatid disease; *post mortem* examination failed to reveal anything else as a possible cause. This point had been debated, but others had had similar cases. In each instance the condition had been hydatid of the liver. A man had worked all day, then suddenly felt sick, vomited and died within a short space of time. A large hydatid cyst had been found occupying the whole of the left lobe of the liver; when the cyst was opened up, hydatid fluid had escaped under considerable pressure. A case had been recorded in Professor Dew's book. Dr. Palmer suggested that death might have been caused by anaphylaxis. There was no obvious escape of blood into the cyst, but under tension hydatid fluid might escape into the blood.

In conclusion, Dr. Palmer referred to the class of cases in which, a careful *post mortem* examination being made, nothing could be found to account for death. It was in such instances that the condition was occasionally pronounced to be *status lymphaticus*.

DR. P. FIASCHI said that he was indebted to Dr. Palmer for his demonstration, and that it was the first time since the war that he had seen anything of the kind. He showed the nose cap of a shell that he had removed at Pozières from under the liver of a soldier. The man was still alive.

DR. A. H. TEBBUTT congratulated the speakers and said that he had enjoyed the chatty informality of their remarks. There was only one point on which he wished to comment. In regard to Dr. Sheldon's reference to cases of *status lymphaticus* (which was more generally called *status thymico-lymphaticus*), he was interested that Dr. Sheldon was so brave as to believe that such a condition existed. There was at the present time a tendency amongst competent people to disbelieve in it. A committee had been appointed by the Medical Research Council of Great Britain to make inquiries into this condition. Dr. Major Greenwood, the statistician, had stated that no such condition existed and that it would be more honest if medical men gave the verdict that the patient had died by act of God. At the last investigation of the Committee it was concluded that an analysis of cases of so-called *status lymphaticus* with the corresponding data (such as weights of thymus *et cetera*) showed that there was no real evidence that there was any greater enlargement of the thymus and thyreoid glands than in similar deaths from unknown causes. It was really not sound to say that death occurred from *status thymico-lymphaticus*. Dr. Tebbutt preferred to believe that the cause of death was not known. He emphasized the necessity for a complete *post mortem* histological examination of all the important viscera. Only thus could such doubt be eliminated and the cases put into known criteria.

DR. E. C. HALL said that, although he had not had the past experience of Dr. Sheldon and Dr. Palmer, he performed the *post mortem* work for the police in a district where the population was 100,000, and had encountered many cases of sudden death. He could confirm what Dr. Sheldon had said in reference to air embolism as a cause of death in cases of attempted abortion. He cited the instance of a young woman at Granville who had been found dead at the bedside with a Higginson's syringe, acetate of lead and Condy's fluid beside her. The vagina contained a large amount of this substance—some had even penetrated to the uterus; there was not much detachment of the placenta. On opening the abdomen a large collection of air bubbles was found in the *vena cava*; there was a similar condition in the heart, the right side being blown out; there was emphysema into the pericardial tissues. Opening of the veins displayed a similar condition. Dr. Hall had never seen such a condition at other *post mortem* examinations. In this instance the examination took place three or four hours after death, so that there was no possibility of decomposition. Dr. Hall therefore confirmed Dr. Sheldon's views.

In regard to rupture of a hydatid cyst, referred to by Dr. Palmer, Dr. Hall said that he had had experience of a similar case. The patient, a man, had been perfectly well; he went to feed a horse in the stable, carrying a tin dish. The horse jammed him against the stall; he collapsed. There were signs of rupture of the viscus; the man refused operation and died shortly afterwards. At *post mortem* examination he was found to have in the abdomen free daughter cysts from a ruptured hydatid cyst.

As to the condition of cerebral hæmorrhage, Dr. Hall again confirmed what Dr. Sheldon had said; it was a common cause of sudden death. In compensation cases, when the cause of death was uncertain, the coroner should be informed and a *post mortem* examination should always be undertaken. He cited the instance of a man who died suddenly while his attending medical practitioner was away. The coroner in charge had a *post mortem* examination performed, and a cerebral hæmorrhage was found at the base of the brain.

Dr. Hall referred to cerebellar hæmorrhage as another cause of sudden death in children. This was not a very common occurrence, and he had only seen one case. *Post mortem* examination revealed the cause of death. The child had been at a carnival; it returned home and was eating bananas; it died in a few minutes. The question of poisoning was raised, but at *post mortem* examination a large cerebellar hæmorrhage was discovered. This would contradict the old idea of cerebral hæmorrhage occurring only in middle aged and old people; it was a cause of death even in the young.

Dr. Hall said that in cases of sudden death it was surprising how often coronary disease was found. It took various forms; he had seen both arteries calcified. Dr. Hall cited another instance of rupture of the coronary artery or coronary vein leading to hæmopericardium. He certainly leaned to the theory that cases of *angina pectoris* might be due either to coronary disease or to coronary occlusion. This was a more satisfactory explanation than any of the other theories.

With regard to *status lymphaticus*, they were on ground that might still be thought very debatable. Not enough was known of the physiology of the thymus gland and of the reasons for its persistence in the adult state in some individuals. In the cases cited by Dr. Sheldon, in the absence of other pathological conditions, death simply had to be ascribed to the enlarged thymus and its accompani-

ments. In epileptics Dr. G. P. U. Prior found that a persistent enlarged thymus was the usual thing. In children it was different. Practically every child with enlarged tonsils and adenoids could be set down as having *status lymphaticus*. When one of these died under an operation for removal of these conditions it was not fair to ascribe it to *status lymphaticus* when it most likely was due to the anæsthetic, such as chloroform, or the spasm of ethyl chloride, or blood in the bronchi leading to asphyxia.

DR. E. L. MORGAN, in regard to the question of *status lymphaticus*, said that he was not aware of the report quoted by Dr. Tebbutt, but Muir's "Text Book of Pathology" described the condition as a definite entity. He quoted a case of sudden death in a patient, aged three, who had been admitted to hospital with a provisional diagnosis of laryngeal diphtheria. There were no signs of diphtheria, but the patient was comatose. Lumbar puncture was performed and a little blood-stained fluid was drawn off. The patient later vomited several times and died in a few hours without regaining consciousness. *Post mortem*, histological examination was performed on portions of the intestine, mesentery, spleen, lung and brain. The notes accompanying the specimens indicated that there was no evidence of laryngeal obstruction, that there was a large pad of adenoid tissue and a persistent thymus, not unduly big, but specimens of these were not submitted for examination. The whole of the lymph glands of the mesentery were enlarged and hyperplastic, as was also the lymphoid tissue of the Malpighian corpuscles of the spleen; the solitary lymph follicles in the intestine stood out like pin-heads; the Peyer's patches were enlarged. The lymphoid tissue everywhere was hyperplastic, without any of the typical features of Hodgkin's disease or lymphosarcoma. In this instance Dr. Morgan thought that the patient died of *status lymphaticus*. Possibly the thymus was pressing on the trachea, thus suggesting diphtheria. Though death was attributed by Muir to heart failure in the great majority of cases, others had suggested anaphylaxis due to abnormal stimulation of the lymphoid tissue following trivial injury, and in this case the trivial injury was supplied by lumbar puncture. The performance of lumbar puncture was interesting, because Muir stated that in these cases the vessels of the brain and the cord might be thin-walled, and minute hæmorrhages were found in sections of the meninges.

DR. E. H. M. STEPHEN spoke of observations made at the Royal Alexandra Hospital for Children, and said that he was satisfied that there was such a state as *status thymo-lymphaticus*. He did not consider that it could be said that deaths under anæsthesia had been proved to be due to *status thymo-lymphaticus*, but subjects of this state were particularly non-resistant to any infection and fared badly however trivial the infection might be.

The pathological department had weighed many thymus glands in patients dying from a variety of causes. The glands had weighed anything from four to thirty-eight grammes.

There had been no uniformity in the results obtained, either according to age or the type of disease responsible for death. Whether it were pneumonia, gastro-enteritis or chronic malnutrition, the gland was sometimes large and sometimes small. As to the appearance of the thymus, it was sometimes long and graceful, sometimes short and stout. The question of the weight of the thymus appeared to have no bearing on the general condition of the child, except in the rare cases of *status thymo-lymphaticus*. Such cases could be identified by the following features: the child was pale, apathetic and flabby, there was often general glandular enlargement and a tendency to rickets, in addition to radiographic evidence of enlargement of the thymus.

Dr. Sheldon, in reply, said for Dr. Palmer's benefit that he had omitted all the doubtful cases in the series, of children, and had confined himself to the thymic deaths in grown-up people.

He did not pretend to offer an explanation of the thymic deaths. He had done hundreds of *post mortem* examinations and had practically never come across persistent thymus glands in grown-up people, except in these and similar cases which had been recorded. The recorded cases were those in which there had been no other evident cause of death. When submitted to the Department of Microbiology nothing particular had been observed, except occasionally hæmorrhages into the thymus. Of course, it would be an excellent thing if the tissues could be submitted to microscopical examination, but he had not the facilities or ability to follow up in this way.

Dr. Sheldon, in submitting his paper, had confined himself more or less to causes of death in which there had been controversy, namely, coronary disease, air embolism and *status lymphaticus* or thymic deaths. He recognized that there were numerous other causes of sudden death than those mentioned by him.

Dr. Palmer said that in going through the index of sudden deaths for the last seventy cases he had been interested to see that 10% of the deaths occurred in people under the age of twenty-five years, and that all of these were from ordinary cerebral hæmorrhage. Of course, it was not suggested that this was the correct percentage, as it was not usual for old people dying from this condition to be brought to the morgue, except in unexpected circumstances.

Correspondence.

WORKERS' COMPENSATION—TYPHOID FEVER.

SIR: On June 13, 1928, I reported to *Public Health* a case at Young, New South Wales, where the widow of a man employed at a nightsoil depôt, who had died from typhoid fever, received £825 compensation from the sanitary contractor of the dry conservancy double pan system, as the result of a judgement under the *Workers' Compensation Act*, New South Wales. At the time the man contracted the fever, there was evidence that there was at least one known case of typhoid fever in the district. In the opinion of the Commission, deceased's employment exposed him to special risks, and the reasonable inference was that the fever which caused his death was contracted in the course of his employment. In my letter I stated this decision must be of great interest. It has generally been believed that employees of nightsoil depôts were singularly free from disease, and in an experience extending over a quarter of a century I do not remember another case where a man engaged in such work contracted typhoid fever. The London Sewer Commissioners, in a report in 1900, commented on the freedom from disease and the good health of sewerage workers.

In *Municipal Engineering and Sanitary Record* (August 9, 1928) the writer of "From the Desk of a M.O.H." commented on this case as follows:

> **A Curious Occupation which is Claimed to be Healthy.**
>
> "No, doctor, George ain't 'alf the man 'e was when he was on the carts. When 'e was on the nightsoil 'e would come in and 'e would 'ave a breakfast; now 'e never seems to enjoy 'is meals, and just mopes about the 'ouse." This, the remark of the wife of a superannuated nightsoil man, came back to me at once on reading the letter of Dr. Purdy, of Sydney, apropos the death of a sanitary depôt employee from typhoid fever, held to have been acquired in the course of his employment.
>
> Like most of us, Dr. Purdy has been reared in the belief that men whose occupation is the emptying of pail closets and the removal and disposal of excreted refuse, are invariably the healthiest of all the employees of a local authority; and that they never by any chance acquire typhoid or, indeed, any other intestinal infection. Also, like most of us, if put to it to find an explanation, he probably has made reference to the possibility of an artificially acquired immunity, the result of repeated small doses of the infection. The times I myself have made such a statement in order to down an opponent of a conversion scheme who has trotted out the healthiness of those who are actually engaged in the handling of pails, are numberless. Commonly, when elaborated, this argument has been attended with a certain amount of success so far as the diehards are concerned, though I am bound to say that I have never been quite successful in satisfying myself of the truth of my contention.

Some Reasons Why It is Healthy.

There is, in fact, an infinity of reasons why these workers should be a strong, healthy and disease-free body. They are, almost without exception, picked men; their work, like their pay, is regular; it is not heavy, it calls for no particular skill, thought or consideration, and, as a body, they are well looked after. There may be, and probably often are, risks of infection with the organisms of typhoid and other intestinal diseases, but having regard to the precautions that are taken on their behalf, they are less than in the case of the occupants of the premises on which the pails are situated. These latter are close up to the concentrated infection, and in the zone where the carriers—flies, blown dust, and so on—are most likely to be active and operative. The chances of precautionary measures, if any, proving ineffective on the ground where the infective material is first deposited are much greater.

In the case of the individuals whose duty it is to collect the material, the precautions necessary are comparatively simple, instinctive almost, and hardly likely to fail. That they must fail once in a while is inevitable; and that they do is shown by this case.

The Exception to the Rule.

Being an exceptional case, Dr. Purdy has done good service in taking the trouble to record it, and very many of us who are so situated, having still areas in which the water carriage system has not yet been fully adopted, that we may find ourselves faced with a similar happening, will note the case for reference at an appropriate moment. Also, it may be hoped, some of us will try to comply with Dr. Purdy's request for information as to other cases. In these days, when typhoid fever is of so comparatively rare occurrence, it should not be difficult to trace the cases in which the victim was a nightsoil worker, but for information as to cases in the past, there must be reliance upon the recollection of those who, without offence, may be called veterans. Classing myself, for the moment, as one of these, I can say quite definitely that I cannot remember one. My own belief has been the orthodox one as to the healthiness and immunity from disease of workers of this class, and nothing has occurred to shake it. If others have had a different experience, I hope they will hasten to put it on record, not only for Dr. Purdy's information, but for that of old-timers amongst their colleagues in this part of the Empire.

The views of British medical officers from the legal aspect are given in *Municipal Engineering and Sanitary Record* (August 9, 1928) as follows:

> Among the numerous letters we have received on the subject from medical officers of health, is one from Dr. Newsholme, the Medical Officer of Health to the City of Birmingham, who writes that he has looked up the records of cases of enteric fever which have occurred in Birmingham during the last ten years, but with the exception of occasional cases among the nursing and domestic staff of the infectious diseases hospital, can find no instance in which the disease attacked any person engaged in other general sanitation or salvage works.
>
> Dr. Ross, the Medical Officer of Health for Whitby, writes that no case of typhoid fever has occurred in Whitby during his tenure of the medical officership.

The medical officer of health who writes the weekly notes "From the Desk of a M.O.H.", furnishes, on page 158 of *Municipal Engineering and Sanitary Record* (August 9, 1928), much informative matter on the subject, remarking that he "cannot remember the occurrence of a case of typhoid fever in an employee of the Sanitary Department".

No Abnormal Sickness at Hull.

Dr. Allen Daley, Medical Officer of Health, Hull, writes:

> There is no record in this department of any abnormal sickness either infectious or non-infectious, amongst the nightsoil workers. Fortunately the number engaged on this work has diminished very rapidly during the last few years, but even at the time when there were large numbers engaged thereon, the statistics were not unsatisfactory. At the present moment our average number of sick of the 225 men and boys engaged on nightsoil collection and dry dust is 8·33. This being 3·70 per cent., is no more than the average amongst any similar number of men engaged in strenuous work.

The Legal Aspect.

The views of English medical officers of health having been thus given, we submit the following letter from a legal authority, which gives a succinct statement of the law on the subject:

Sir: Referring to Dr. Purdy's interesting letter under the above heading in your issue of the 26th ultimo, at page 93, the following three legal decisions may be mentioned as having some bearing on the question he asks:

(1) In the case of Finlay *v.* the Guardians of the Tullamore Union (1914), 2 Irish Reports, 233, a workman was employed as a machinery attendent at certain sewage pumping works, and part of his work consisted in removing sewage out of the machinery. After having been engaged several years in this employment, he contracted typhoid fever, of which he died. Medical evidence was given that he might have acquired the disease by handling the sewage, but no evidence was given that the removal of the sewage was, in fact, the cause of his death. The court, after a full hearing, held that the evidence did not establish that death resulted from an accident in the course of his employment, and that there was no right of compensation under the *Workmen's Compensation Act*, 1906.

(2) In Broderick *v.* the London County Council (1908), 2 K.B., 807, the County Court Judge found as a fact that a workman employed in the County Council's sewers had contracted enteritis—a kindred disease to typhoid, as we understand the term—from inhaling sewer gas. The Court of Appeal, however, whilst not challenging this finding, held that the case was not one of "injury by accident" within the meaning of the *Workmen's Compensation Act*.

(3) In the case of Eke *v.* Hart-Dyke (1910), 2 K.B., 677, the County Court Judge found as a fact that a man who had been employed for some four or five days in opening out certain cesspools for inspection, died from the results of poisoning, contracted whilst working on the cesspools. Here again the Court of Appeal held that this did not constitute an "injury by accident" so as to entitle the man's widow to compensation under the above Act.

A second case came before the Workers' Compensation Commission in Sydney on November 3, 1931, of which the following is the report in the Sydney *Sun*.

Worker Gets Enteric.

That Thomas Spencer Foley, an employee of the Water Board, had contracted enteric fever in the course of his employment, was decided by the Workers' Compensation Commission today, and an award in his favour was made of £5 per week from December 26, 1930, till May 17, 1931—£4 17s. a week from May 18 till September 7, 1931, and £2 8s. 6d. a week from 8th September till 19th October, 1931. Foley was tunnelling a trench for a sewer underneath ordinary household mains which were "alive" at the time, and frequently had to move pipes containing foul matter. Through enteric he had become totally incapacitated.

Dr. Egan, who was the medical attendant of the plaintiff, attended him from the beginning of his illness, and in evidence stated that in his opinion the disease was associated with his occupation and contracted in connexion therewith.

Under cross examination as to the possibility of infection from milk, he pointed out that the patient

was emphatic that he never took milk, even in his tea, and, moreover, there was no other case associated with the milk supply within that neighbourhood.

The Sydney water supply could not be at fault, since this would mean an explosive outbreak.

The eating of oysters was denied by the plaintiff also; he had not eaten any lettuce or any food from which there was a risk of contamination.

Dr. Egan stated that as he lived in the neighbourhood of the sewers, he had personally seen the plaintiff working in the sewer, where there were fæces, and he had seen sewer workers actually rolling cigarettes with hands contaminated with fæces.

An interesting feature with regard to the incidence of typhoid fever in Sydney was that actually for the last six months of 1930 there were 26 cases in the sewered portion of the metropolitan area and 13 in the non-sewered portion. The population of the metropolitan area on December 31, 1930, was 1,333,840. The number of houses connected to the sewer was 204,177, serving a population estimated at 1,020,885. On June 30, 1930, the population in the unsewered area was 323,355. It will be seen therefore that nearly a quarter of the population in Sydney has not yet the advantage of sewerage for their dwellings. The incidence per 100,000 was 0·2 in the sewered and 0·4 in the unsewered area.

As a matter of fact, one had urged repeatedly, in season and out of season, that the extension of the sewerage system was a work that should have been pushed on with the certitude of an adequate return in improved health and comfort. One felt ashamed recently when a large factory was erected in one of our large industrial suburbs by a Granville firm, that it was neither possible to connect with the sewerage system nor instal a septic tank. With regard to the latter expedient, little effort was made by the tenants of the adjoining estate to cooperate.

Although we live in the third decade of the twentieth century and the care of the public health is considered to be the first concern of a statesman, it cannot be said that the concern has been interpreted by intensive action as regards the extension of sewerage in Sydney, such not having been proportionate to the marked increase of population in recent years.

"It never rains but it pours." It is remarkable that, although able to state that in an experience of thirty-three years I had never known another case of typhoid fever in a sewer worker or a nightsoil contractor's employee, other than the case at Young and the last case, within a few weeks another case, that of a man who had been engaged in the removal of nightsoil in a dry conservancy system, should have arisen.

Two cases of typhoid in employees at Lila Springs, who cleaned out pit latrines in 1926, are recorded in the Workers' Compensation Reports.

One feels warranted, therefore, in suggesting that all sewer workers and people engaged in the removal and burial of human excreta, should be protected by immunization, either by antityphoid inoculation—which, of course, would not be popular, as no one seems to like getting the needle—or by oral administration of vaccine.

The South African Council of Public Health, 1929, recommended prophylactic immunization of all contacts, and emphasized the advantage in certain circumstances of the oral method of immunization. It is claimed that immunity is established more quickly by the oral than by the injection method. Many persons who object to injections, are willing to take medicine in pill or tablet form by the mouth. The vaccine is quite harmless and its action is aided by ox-bile tablets taken last thing at night and a vaccine tablet first thing in the morning for three successive days. The South African Institute for Medical Research, Johannesburg, provides the tablets to local authorities at a cost of ninepence per dose for one person (that is, three bile and three vaccine tablets).

Such immunization, whether by oral or injection method, is not in any way a substitute for sanitary measures to prevent the occurrence and spread of typhoid infection, but is regarded merely as an immediate and supplementary precaution.

Such prophylaxis against typhoid fever is to my mind a logical corollary of what is shown to be an actual risk, but which has hitherto not been entertained as probable on account of insufficient data having been collected.

Yours, etc.,

J. S. PURDY,

Metropolitan Medical Officer of Health.

Sydney,
January 19, 1932.

PLANTS INJURIOUS TO MAN IN AUSTRALIA.

SIR: Might I be permitted to add some observations to Professor J. Burton Cleland's communication in your issue of December 19, 1931?

The stinkwort (*Inula graveolens*): I had brought under my notice in connexion with the *Workers' Compensation Act*, in April last, three members of one family suffering from *dermatitis venenata* caused by this weed, in the Orange district of New South Wales. The father was aged forty-seven and the two sons respectively twenty-two and eighteen. The face in each case was markedly affected, pruritus was intense and constitutional symptoms were present. The periods of incapacity were eight, twelve and fourteen days. This imported weed was first introduced into South Australia about 1863 and has since spread to Western Australia, Victoria and New South Wales. The plant was known to the ancient Greeks. It possesses a volatile oil and has been used for colic, dysuria, amenorrhœa and asthma. Pharmacological experiments on animals produced respiratory paralysis and locomotor disorder. Apparently the plant contains two constituents, one of which convulses the anterior limbs and the other paralyses the hind limbs, the latter effect predominating. Stinkwort is closely related to the well-known elecampane (*Inula helenium*).

Milk-bush (*Sarcostemma australe*): The veteran botanist, Mr. Fred Turner, F.L.S., has reported that in north-western New South Wales the aborigines applied the milky juice of the plant to wounds *et cetera*. He has also stated that the native blacks used an infusion of the caustic plant for dysentery. In North Australia the aborigines at Darwin employed the juice as a smallpox remedy. The late J. H. Maiden quoted a Mrs. Kennedy, of Wonnaminta, in western New South Wales, as authority for a statement that the juice was a very valuable remedy, rapidly curing corns and warts. The use of the milky juice as a healing application to wounds is well known to settlers. The caustic vine, however, is suspected of being poisonous to stock at certain seasons of the year.

As regards fly-killing plants, *Lomatia silaifolia* (Proteaceæ) has a great reputation. This plant is termed wild parsley from the appearance of its foliage. Vases containing the flowers are often surrounded by dead flies. It is stated that the pollen contains a small quantity of a glucoside yielding hydrocyanic acid.

Grass seeds may not only injure the fleeces and skins of sheep, but may also get into the conjunctival sac in numbers and set up inflammatory changes leading to blindness. When in the country I have removed "black oats" from the conjunctival sac, nasal cavities and external auditory meatus of human subjects.

Yours, etc.,

JOHN MACPHERSON.

Macquarie Street,
Sydney,
January 8, 1932.

Obituary.

FREDERICK CHARLES RORKE.

WE regret to announce the death of Dr. Frederick Charles Rorke, which occurred at Gundagai, New South Wales, on February 7, 1932.

Books Received.

TOWARDS NATIONAL HEALTH, OR HEALTH AND HYGIENE IN ENGLAND FROM ROMAN TO VICTORIAN TIMES, by J. A. Delmege, O.B.E., M.R.C.S., L.R.C.P., D.P.H., with a foreword by Sir Thomas Legge; 1931. London: William Heinemann (Medical Books). Crown 4to., pp. 248, with illustrations. Price: 21s. net.

HYDROTHERAPY AND PHYSIOTHERAPY FOR BATH ATTENDANTS, NURSES AND BIOPHYSICAL ASSISTANTS, by L. C. E. Calthrop, M.B., M.R.C.S., L.R.C.P.; 1931. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 184, with illustrations. Price: 5s. net.

AN INDEX OF PROGNOSIS AND END-RESULTS OF TREATMENT, by Various Writers; Edited by A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; Fourth Edition; 1932. Bristol: John Wright and Sons, Limited. Imperial 8vo., pp. 610. Price: 2 guineas net.

THE CONQUEST OF OLD AGE, METHODS TO EFFECT REJUVENATION AND TO INCREASE FUNCTIONAL ACTIVITY, by Peter Schmidt, M.D., translated by E. and C. Paul; 1931. London: George Routledge and Sons Limited; Sydney: Moore's Bookshop. Royal 8vo., pp. 314, with 40 full-page plates. Price: 21s. net.

EMERGENCY SURGERY, by J. W. Sluss, A.M., M.D., F.A.C.S., and J. W. Martin, M.D., F.A.C.S., assisted by D. H. Sluss and C. B. De Motte; Fifth Edition; 1931. Philadelphia: P. Blakiston's Son and Company. Demy 8vo., pp. 894, with 797 illustrations, some in colour. Price: $5.00 net.

Diary for the Month.

FEB. 23.—New South Wales Branch: Medical Politics Committee.
FEB. 24.—Victorian Branch: Council.

Medical Appointments.

Dr. H. McI. Birch (B.M.A.) has been appointed Acting Deputy Superintendent, Parkside Mental Hospital, South Australia.

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Dr. G. H. Moore (B.M.A.) has been appointed Acting Chief Quarantine Officer (General), New South Wales, pursuant to the provisions of the *Quarantine Act*, 1908-1924.

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Dr. H. T. Bourne (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane and Receiving House, Royal Park, Victoria, pursuant to the provisions of the *Lunacy Act*, 1928.

* * *

Dr. A. R. Southwood (B.M.A.) has been appointed Chairman of the Central Board of Health and Vaccination Officer, and Chairman of the Advisory Committee under the *Food and Drugs Act*, 1908, Division II, under the provisions of *The Public Service Acts*, 1916 *to* 1925, South Australia.

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Dr. J. W. Rollison (B.M.A.) has been appointed Deputy Superintendent, Northfield Mental Hospital, Medical Officer, Yatala Labor Prison, and Medical Officer, Northfield Consumptive Home, to be also Assistant Honorary Medical Officer, T.B. (*sic*) Clinic, North Terrace, Adelaide, South Australia, under the provisions of *The Public Service Acts*, 1916 *to* 1925.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Honorary Officers.
HOBART PUBLIC HOSPITAL, TASMANIA: Resident Medical Officers.
MATER CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: House Physician, House Surgeon.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Dermatologist, Honorary Assistant Physician.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Research Assistant.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chilliagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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